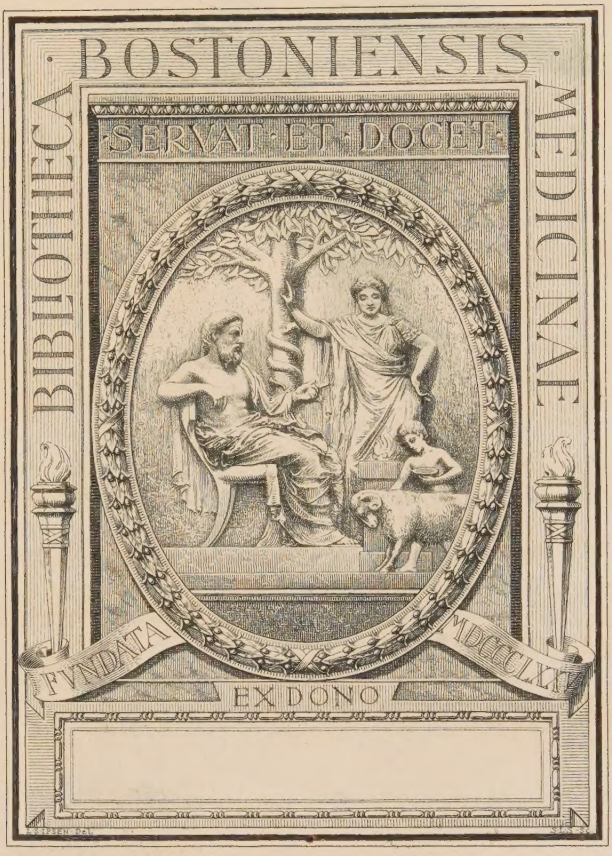
HC 4QE3 X

28.A.412


BIBLIOTHECA · BOSTONIENSIS · MEDICINAE
SERVAT · ET · DOCET
FVNDATA · MDCCCLXXI
EX DONO

Digitized by the Internet Archive
in 2025

https://archive.org/details/diagnosisfromocu0000matt

DIAGNOSIS FROM OCULAR SYMPTOMS

DIAGNOSIS

FROM

OCULAR SYMPTOMS

BY

MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S.

MEMBER OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY; OPHTHALMIC SURGEON TO THE NEW ROCHELLE HOSPITAL; FIRST LIEUTENANT IN THE MEDICAL RESERVE CORPS, UNITED STATES ARMY

NEW YORK

REBMAN COMPANY

141 West 36th Street

BOSTON MEDICAL
APR 20 1917
LIBRARY

14379

COPYRIGHT, 1917, BY
REBMAN COMPANY
NEW YORK

All Rights reserved

PRINTED IN AMERICA

This Work is Dedicated

TO THE MEMORY OF

MY FATHER

GEORGE TIFFANY FOSTER, M.D.

AN OBSCURE COUNTRY PRACTITIONER

OF RARE DIAGNOSTIC ACUMEN

TABLE OF CONTENTS

PAGE

Introduction 1

CHAPTER I

Symptoms, History Taking, and Differentiation 5
Oblique or Focal Illumination 7
Ophthalmoscopy 7
The Indirect Method 9
The Direct Method 10
History Taking 11
Differentiation 13

CHAPTER II

The Lids 15
Epicanthus 15
Ptosis 16
Congenital Ptosis 16
Acquired Ptosis 17
Winking 23
Blepharospasm 24
Œdema of the Lids 26
Emphysema of the Lids 28
Ecchymoses of the Lids 28
Hyperæmia of the Skin of the Lid 29
Eruptions and Discolorations on the Lids 29
Herpes Zoster 30
Discolorations 31
Atrophy of the Skin of the Lid 31
Hypertrophy of the Skin of the Lid 32
Disorders of Secretion of the Skin of the Lid 32
Sores on the Lids 32
Tumors of the Lid 35
Blepharophimosis 40
Lagophthalmos 41
Ectropion 41
Entropion 43
Spastic Entropion 44
Cicatricial Entropion 44
The Eyelashes 45
Trichiasis 46

PAGE

Blepharitis Marginalis 46
Hyperæmia of the Margin of the Lid 47
Seborrhea 47
Blepharitis Ulcerosa 48
Leprosy 50

CHAPTER III

The Lacrimal Organs, Lymphatic Glands, and Accessory Sinuses 51
The Lacrimal Organs 51
Dacryocystitis 53
Lacrimal Fistulæ 55
Dacryoadenitis 56
Dacryops 57
The Lymphatic Glands 58
The Accessory Sinuses 59

CHAPTER IV

Displacements of the Eyeball 62
Enophthalmos 62
Exophthalmos 63
Exophthalmic Goitre 65
Hemorrhage into the Orbit 66
Emphysema of the Orbit 66
Luxation of the Eye 67
Exophthalmos Due to Syphilis and Tuberculosis 67
Exophthalmos Caused by Tumors 68
Pulsating Exophthalmos 71
Exophthalmos Caused by Vascular Trouble 72
Inflammatory Exophthalmos 73
Orbital Cellulitis 73
Orbital Periostitis 74
Exophthalmos Due to Tenonitis 76
Exophthalmos Caused by Cerebral Disturbances 76

CHAPTER V

Deviations of the Eyeball 78
Differentiation of Strabismus and Muscular Paresis 79
Strabismus 82
Causes of Convergent Strabismus 82
Causes of Divergent Strabismus 85
Observations to be made in Strabismus 86

PAGE
Muscular Paresis 88
Differentiation of Lesions Productive of Paresis of the Ocular Muscles 94
Peripheral Pareses 95
Nuclear Pareses 96
Conjugate Paresis 99
Paralysis of Convergence 100
Paralysis of Divergence 100

CHAPTER VI

Motor Anomalies without Deviation of the Eyeballs 101
Heterophoria 102
Cyclophoria 105
Nystagmus 106

CHAPTER VII

The Conjunctiva 110
The Caruncle 111
The Semilunar Fold 112
Congenital Defects of the Conjunctiva 112
Pinguecula 112
Spots on the Conjunctiva 113
Pterygium 113
False Pterygium 114
Tumors of the Conjunctiva 114
Cysts of the Conjunctiva 115
Symblepharon 116
Xerosis 116
Subconjunctival Hemorrhage 117
Emphysema of the Conjunctiva 118
Chemosis 118
Redness of the Eyeball 119
Anæmia of the Conjunctiva 120
Hyperæmia of the Conjunctiva 121
Chronic Conjunctivitis 121
Diplobacillus Conjunctivitis 122
Foreign Body on the Conjunctiva or Cornea 122
Acute Catarrhal Conjunctivitis 124
Squirrel Plague Conjunctivitis 127
Ophthalmia Nodosa 127
Gonorrheal Conjunctivitis 128
Gonorrheal Conjunctivitis of Adults 128
Metastatic Gonorrheal Conjunctivitis 129
Gonorrheal Conjunctivitis of Infants 130
Membranous Conjunctivitis 131

PAGE
Vernal Catarrh 134
Trachoma 136
Trachomatous Pannus 139
Sequelæ of Trachoma 140
Benign Follicular Affections of the Conjunctiva 141
Follicles Induced by Conjunctivitis 142
Follicles Induced by Drugs 143
School Folliculosis 143
Follicular Conjunctivitis 144
Tuberculosis of the Conjunctiva 146
Parinaud's Conjunctivitis 148
Phlyctenular Conjunctivitis 149
Herpes Simplex 150
Pemphigus 151
Syphilitic Lesions of the Conjunctiva 151
Amyloid and Hyaline Degenerations of the Conjunctiva 153
Calcification of the Conjunctiva 153
Leprosy of the Conjunctiva 154
Burns of the Conjunctiva 154

CHAPTER VIII

The Cornea 156
Irregular Astigmatism 156
Keratoconus 157
Keratoglobus 158
Anæsthesia of the Cornea 158
Pigmentation of the Cornea 159
Opacities of the Cornea 161
Wounds, Foreign Bodies, and Abrasions of the Cornea 162
Burns of the Cornea 163
Ulcers of the Cornea 164
Nebula, Macula, Leucoma 166
Arcus Senilis 167
Degenerative Changes in the Cornea 167
Keratocele 168
Perforation of an Ulcer and its Consequences 168
Ectasia of the Cornea 169
Staphyloma of the Cornea 170
Ulcers of the Cornea in Infancy 171
Ulcers of the Cornea in Childhood 172
Phlyctenular Keratitis 172
Ulcers of the Cornea in Middle Life and Old Age 174
Mooren's Ulcer 174
Serpiginous Ulcer 174
Ulcers and Inflammations of the Cornea Met with at All Ages... 177
Ulcers Due to Fungi 177
Keratitis e Lagophthalmo 177

PAGE
Neuroparalytic Keratitis 179
Dendritic Keratitis 180
Febrile Herpes of the Cornea 181
Herpes Zoster Ophthalmicus 182
Bullous Keratitis 183
Filamentary Keratitis 184
Superficial Punctate Keratitis 185
Interstitial Keratitis 185
Abscess of the Cornea 190
Striped Keratitis 191
Deep Punctate Keratitis 191
Sclerosing Keratitis 191
Keratomalacia 192
Tumors of the Cornea 192

CHAPTER IX

The Sclera and the Anterior Chamber 194
The Sclera 194
Pigmentations of the Sclera 194
Staphyloma of the Sclera 194
Scleritis 195
Episcleritis 197
Episcleritis Periodica Fugax 198
The Anterior Chamber 198
Obliteration of the Anterior Chamber 199
Shallow Anterior Chamber 199
Deep Anterior Chamber 200
Visible Changes in the Aqueous 200

CHAPTER X

The Iris 202
Persistent Pupillary Membrane 203
Color of the Iris 203
The Normal Pupil 204
Reactions of the Pupil 205
Reaction of the Pupil to Light 205
Reaction of the Pupil to Convergence 206
Myotonic Convergence Reaction 207
Reaction of the Pupils to Closure of the Lids 207
Cortical or Attention Reflex of the Pupils 207
Reflex Dilatation of the Pupil 208
Malformations of the Pupil 208
Posterior Synechiæ 209
Oval Pupil 209
Corectopia 209
Polycoria 210

PAGE

Iridodonesis 210
Iridodialysis 211
Inversion of the Iris 211
Irideremia 211
Coloboma of the Iris 213
Iritis 215
Hyperæmia of the Iris 216
Serous Iritis and Cyclitis 216
Plastic Iritis 218
Ætiology of Iritis 219
Secondary Iritis 224
Traumatic Iritis, Cyclitis, and Iridocyclitis 225
Iritis Met With in Acute Infectious Diseases 225
Syphilitic Iritis 226
The Papular Form of Syphilitic Iritis 228
Gummatous Iritis 230
Tuberculosis of the Iris 230
Gonorrheal Iritis 231
Gouty Iritis 232
Rheumatic Iritis 233
Iritis from Anæmia 234
Purulent Iritis 235
Atrophy of the Iris 236
Tumors of the Iris 236
The Symptomatology of the Pupils 237
Mydriasis 237
Myosis 238
Anisocoria 240
Hippus 241
Alternating Mydriasis 242
Hemianopic Pupillary Reaction 242
Slowness of Reaction of the Pupil to Light 243
Amaurotic Immobility of the Pupil 243
Absolute Immobility of the Pupil 244
Reflex Immobility of the Pupil 245

CHAPTER XI

The Lens 247
Dislocation of the Lens 247
Luxation and Subluxation 247
Ectopia 249
Coloboma of the Lens 251
Lenticonus 251
Cataract 252
Congenital Cataract 253
Zonular or Lamellar Cataract 253
Total Cataract 254
Central Cataract 254
Fusiform or Spindle Cataract 255

PAGE
Posterior Polar Cataract 255
Anterior Polar Cataract 256
Punctate Cataract 256
Degenerated Cataract 257
Traumatic Cataract 257
Cataract Due to Disease 261
Senile Cataract 264

CHAPTER XII

The Vitreous 272
Remains of the Fetal Hyaloid Artery 272
Muscæ Volitantes 273
Fluid Vitreous 274
Hyalitis 275
Hemorrhage into the Vitreous 276
Recurrent Hemorrhages into the Vitreous 277
Preretinal Hemorrhages 277
Foreign Bodies and Parasites in the Vitreous 278

CHAPTER XIII

Abnormal Tension of the Eyeball 279
Glaucoma 280
Prodromal Symptoms of Glaucoma 281
Acute Inflammatory Glaucoma 282
Chronic Inflammatory Glaucoma 284
Simple Glaucoma 285
Infantile Glaucoma 287
Secondary Glaucoma 288
Hemorrhagic Glaucoma 290
Absolute Glaucoma 291
Subnormal Tension 291

CHAPTER XIV

Injuries of the Eye, and Sympathetic Ophthalmia 294
Contusions 294
Contusions without Rupture of the Capsule of the Eye 294
Contusions with Rupture of the Capsule of the Eye 300
Penetrating Wounds of the Eye 301
Foreign Bodies in the Eye 302
Infection of a Wounded Eye 305
Panophthalmitis 306
Infection of the Anterior Segment 306
Abscess of the Vitreous 307
Inflammatory Changes Induced by Wounds 307

PAGE

Sympathetic Ophthalmia 308
Sympathetic Irritation 309
Sympathetic Inflammation 310

CHAPTER XV

A General Consideration of the Fundus 314
The Papilla 315
The Margin of the Papilla 316
The Physiological Excavation of the Papilla 317
Differentiation of Physiological from Pathological Excavations 318
The Vessels of the Papilla and Retina 319
Pigmentation of the Papilla 321
The Background of the Fundus 322
The Macula Lutea 323
Light Reflexes 324
Abnormalities of the Fundus 324

CHAPTER XVI

The Papilla 326
Coloboma of the Optic Nerve Sheath 326
Persistent Hyaloid Artery 327
Optic Atrophy 327
Disease of the Papillomacular Bundle of Nerve Fibers 328
Neuritic Optic Atrophy 329
Simple Optic Atrophy 329
Sudden Pallor of the Papilla 330
Gradually Developing Pallor of the Papilla 330
Hereditary Optic Atrophy 332
Redness of the Papilla 333
Optic Neuritis 335
Choked Disk 336
Diagnostic Value of an Optic Neuritis 337
Diagnostic Value of Choked Disk 339
Choked Disk with Tumors of the Brain 340
Choked Disk in Other Forms of Intracranial Trouble 343

CHAPTER XVII

The Retina and Choroid 344
Anæmia of the Fundus 344
Hyperæmia of the Fundus 345
Spots in the Fundus 346
Choroidal Hemorrhages 346
Retinal Hemorrhages 347

PAGE

Small White Spots 348
Gunn's Dots 349
Metallic Dots 349
Colloid Formations 349
Punctata Albescens 349
Inflammatory White Spots 350
The Retinal Vessels 350
Angioid Streaks in the Retina 353
Striate Retinitis 353
Sclerosis of the Retinal Vessels 354
Large White Spots in the Fundus 356
Medullated Nerve Fibers 356
Sclerosis of the Choroid 357
Defects about the Papilla and the Macula Lutea 358
The Halo of the Papilla 358
Conus 358
Posterior Staphyloma 359
Inferior Conus 361
Coloboma of the Choroid 361
Coloboma of the Macula 363
Rupture of the Choroid 364
Proliferating Retinitis 365
Œdema of the Retina 366
Traumatic Œdema of the Retina 367
Hole in the Macula 367
Occlusion of the Central Artery of the Retina 368
Occlusion of a Branch of the Central Artery 370
Retinitis 370
Central Retinitis 372
Septic Retinitis 374
Albuminuric Retinitis 374
Albuminuric Retinitis of Pregnancy 375
Diabetic Retinitis 376
Syphilitic Retinitis 376
Saturnine Retinitis 377
Anæmic Retinitis 378
Leucocythæmic Retinitis 378
Gouty Retinitis 378
Hemorrhagic Retinitis 378
Thrombosis of the Central Vein 379
Pigmentary Degeneration of the Retina 380
Amaurotic Family Idiocy 383
Maculocerebral and Macular Degenerations 383
Circinate Degeneration 386
Choroiditis 386
Central Choroiditis 390
Senile Degeneration of the Macula 391
Disseminated Choroiditis 391
Tuberculous Choroiditis 393
Purulent Choroiditis 394

PAGE
Glioma of the Retina 395
Detachment of the Retina 399
Detachment of the Choroid 404
Tumors of the Choroid 405

CHAPTER XVIII

Amblyopia, Affections of the Color Sense, and Defects of the Visual Field 408
Amblyopia 409
Congenital Amblyopia 409
Acquired Amblyopia 410
Traumatic Amblyopia 410
Amblyopia from Bright Light 410
Amblyopia from Disease 411
Reflex Amblyopia 411
Hysterical Amblyopia 412
Toxic Amblyopia 413
Toxic Amblyopia from Quinine 414
Toxic Amblyopia from Alcohol and Tobacco 415
Wood Alcohol Poisoning 415
Amblyopia from Overwork 416
Malingering 417
Affections of the Color Sense 420
Chromatopsia 420
Color Vision 421
Color Blindness 422
Detection of Color Blindness 424
Defects in the Field of Vision 426
The Normal Field of Vision 426
Tests of the Visual Field 426
Measurements of the Field of Vision 427
Contraction of the Field of Vision 428
Sectorshaped Defects in the Field of Vision 430
Scotomata 430
The Blind Spot 431
Central Scotoma 431
Ring Scotoma 431
Scintillating Scotoma 432
Hemianopsia 432

CHAPTER XIX

Headache, Neuralgia, and Eyestrain 437
Facial Neuralgia 438
Eyestrain 440
Eyestrain Due to Refractive Errors 443

PAGE

Presbyopia 446
Hypermetropia 447
Myopia 452
Astigmatism 453
Eyestrain Due to Heterophoria 454
Eyestrain Due to Causes Outside of the Eye 457
Nervous Asthenopia 458
Nasal Asthenopia 461

CHAPTER XX

Psychical Symptoms Associated with the Sense of Vision .. 467
Psychical Blindness 467
Visual Hallucinations 468
Conditions in Which Visual Conception Seems to be Dissociated from Other Mental Faculties 468
Visual Aphasia 469
Visual Amnesia 469
Alexia 469
Amnesic Color Blindness 470
Dyslexia 470

INDEX 471

DIAGNOSIS FROM OCULAR SYMPTOMS

INTRODUCTION

Many of the troubles that lead patients to visit us at our offices are self limited and will get well of themselves if let alone, but others, which perhaps resemble them closely, are not so obliging, and, if not rightly treated, will go on to impairment of the vision, destruction of the eye, or possibly loss of life. Right treatment must be based on a correct diagnosis. This is plain to the patient himself, who is justified in asking us if we know just what is the matter with him before he begins to follow our directions. Snap diagnoses are brilliant sometimes, but they are quite as apt to be wrong, and are hazardous even when they are based on resemblances to previous cases that have been carefully studied. The most experienced diagnosticians are the least prone to make snap diagnoses, and are the most apt to study symptoms closely. These are truisms that apply to every branch of medicine. The specialist meets with some of the diseases of the eye every day, others he has never seen, and the more experienced he is the more constantly is he on the qui vive lest he should make a mistake. The danger of error increases with the lack of studious experience and is greatest among practitioners whose patients have yet to arrive, and those who meet with only a small percentage of eye cases. These find it extremely difficult to arrange the symptoms presented by a patient so as to arrive at a diagnosis by exclusion. It is not uncommon for a serious condition in the eye or orbit to be overlooked by a physician whose attention has been caught and held by some other, perhaps less important, lesion or symptom elsewhere in the body, as when a case of glaucoma is mistaken for one of trigeminal neuralgia. Literature abounds in excellent descriptions of individual diseases which we study in the hope that we shall recognize them when they appear before us, but their number is so great, and the resemblances some bear to others

are so close, that memory cannot be relied upon to differentiate one from another. We cannot make a diagnosis by exclusion through calling up mental pictures of every possible disease which may present certain symptoms, and then eliminating all that do not correspond in every particular to the clinical picture presented by the individual patient. We must gather together all of the symptoms that can be obtained, select the prominent ones which indicate the group of diseases to which this one belongs, and narrow the group more and more by an orderly consideration of the remaining symptoms until but one disease is left. Then we have made a diagnosis by exclusion, but the process is not as easy as it sounds.

Ever since entering upon the study of ophthalmology I have wished that some bright mind would analyze the symptoms that appertain to or appear in the eye, select certain ones for points of departure, and arrange the others into syndromes, showing how those which resemble each other differ, and how exclusion is to be made. A colossal dream perhaps, certainly a stupendous task, yet one well worth undertaking, if only to make a single step toward rendering accurate diagnosis easier to the unskilled. Many books have been written, many beautiful plates published that are of immense help in diagnosis, but all seem to illustrate individuals, to introduce them as it were, without pointing out with sufficient clearness the distinguishing marks by which each could be differentiated from all others. Finally I undertook the task, but with many misgivings, for I have no claim to be possessed of surpassing ability as a diagnostician. The completed work falls far short of my ideal. I can only hope that it contains a suggestion as to presentation which will be developed better in the future.

The system adopted is to try to utilize the symptom or condition that predominates in the mind of the examiner as the central figure from which to work outward toward the diagnosis, to collate about this the possibilities with the characteristics of each, and then to discuss individual diseases sufficiently to make each picture clear. No attempt is made to fill in all of the details, because a multiplicity of symptoms that are adventitious rather than diagnostic seems to me to be confusing. Once the diagnosis has been reached the details can be filled in and the result compared with the complete and beautiful delineations that abound. Brevity is employed for the sake of clearness, not to save space, and the omission of any important diagnostic points must be attributed to my lack of appreciation of their

importance. At the same time omissions and errors have been found and corrected even on the last reading of the manuscript. Probably some remain, but it is hoped that the number is not large. Ætiology and course frequently are valuable diagnostic aids, and are included as such. Frequent reference is made to the inestimable assistance furnished by pathology, bacteriology, and biological research, but the details of the methods by which such help is obtained are omitted, because to deal with them would carry us far afield of our subject. Treatment is outside of the scope of this work. Numerous repetitions and cross references have been made necessary by the realization that when a patient is examined by two or more physicians a different symptom may be the more strongly impressed on the mind of each, so that each will have a different point of departure from which to construct his diagnosis, and also by the fact that a symptom which predominates in one case may appear to be subordinate in another. The symptomatology of diseases of other parts of the organism is gone into only in so far as it seemed necessary for the understanding of the associated ocular symptoms. My only reply to a critic who claims that this is not a system, but an unscientific lack of one, is that if he can devise anything better suited to meet the needs of physicians who are intent on finding out what is the matter with their patients, he should do so at once and relegate this poor attempt to the background. The great question is whether this book will enable students to diagnose diseases in which the eye is affected more readily and correctly than others which they have at command, or not, and the answer can come only from the students themselves.

Wherever I have consciously appropriated the ideas of some one else, I have tried to give credit for them, but in many cases the source of the ideas has been forgotten. Probably I am indebted to *Roemer* more than to any other one author, because of my familiarity with his textbook, but I am under obligation to a very large number of writers, the names of many of whom will be found scattered through the volume.

The absence of illustrations deserves an explanation, as the publisher has very kindly put a large number of plates at my disposal and urged their acceptance, but after a long period of consideration I have decided not to avail myself of the courtesy. My endeavor is to present pen pictures that bring out strongly the points through which differentiation is made, and it is practically impossible to pre-

sent these points in a plate so as to make clear the differentiation between two conditions that closely resemble each other. Illustrations in black and white are little more than suggestive; we are not apt to recognize them unless we know what they are intended to represent, and then our memories and imaginations supply the deficiencies. No one can admire more than I do the perfection of reproduction to be seen in the best colored plates, but these seem to me to place too strong an emphasis on what is simply visible, and they necessarily portray the details which are peculiar to the one individual case as distinctly as those from which aid is obtained in making the diagnosis. They seem to be easier to study than pen pictures, yet if anyone doubts the correctness that they place too great emphasis on certain of the diagnostic symptoms, let him take an atlas of the very best, with which he is not acquainted, cover up the titles, and try to diagnose the conditions portrayed; his surprise when the titles are uncovered may be illuminating. From such a plate we can perceive that the lids are swollen, that the conjunctiva is inflamed, there is an ulcer of the cornea, that one eye deviates or protrudes, or that there are certain gross lesions in the fundus, but these are only steps toward the diagnosis. We need to learn the cause of the swollen lid, and of the conjunctivitis. No one can differentiate through such a picture a pneumococcal from a diplococcal ulcer of the cornea, a strabismus from a muscular paresis, the cause of an exophthalmos, or an albuminuric from one of many other forms of retinitis, yet these are the important things to be determined in each individual case. Furthermore, such plates seem to me to distract attention from the text, and to lead the beginner to look for duplicates in his practice, which are seldom to be found. For these reasons I have decided to do without such illustrations, although I appreciate their beauty and attractiveness.

CHAPTER I

Symptoms, History Taking, and Differentiation

When we speak of a **symptom** we refer to some abnormality in the tissue or the function of the eye, which may be perceptible to the patient himself, to an observer, or to both. We call it subjective when it is appreciable by the senses of the patient alone, objective when it can be perceived by another person. Pain, dizziness, and dimness of vision are subjective symptoms, while a swollen lid, a spot in the fundus, an abnormal tension of the eyeball, and a pulsation of either a blood vessel or of the eyeball are objective. Broadly speaking the findings of pathological, bacteriological, and biological findings are objective symptoms, because we gain a knowledge of them through the senses of others than the patient. With extremely rare exceptions a single symptom of either nature standing alone gives us no clue to the site or to the nature of the trouble which produced it, but simply indicates that something is wrong somewhere. Severe pain in the eye may be caused by an inflammation of some of the tissues of that organ, by increased intraocular tension, by a refractive or muscular error, or by a fault in some other organ, like the nose or the stomach, and a swelling of the lid may likewise be a sign of a local inflammation, or of disease in a far distant part, like the kidney. The presence of one symptom leads us to look for others, which usually can be found, but when they cannot, the diagnosis seldom can be made, although it may be suspected, as when a patient has a reflex immobility of both pupils, but no other signs of tabes. In the great majority of cases we can trace a causal relationship between a subjective and an objective symptom, as when an impairment of vision is caused by an opacity in the media, but this is not invariably the case. In one case almost all of the symptoms may be subjective, as in eyestrain from heterophoria, in which the only objective symptom obtainable may be a movement of the eye in the cover test, while in another they may be wholly objective, as when a commencing sclerosis of the retinal vessels, or a small coloboma of the lens affects the

function of the eye in no way, so far as can be determined. Occasionally we can demonstrate the presence of a subjective symptom of which the patient is unaware, as when we find a scotoma in the peripheral portion of the field, which may correspond to a lesion in the fundus, but evades his direct observation.

It is natural that we should have more confidence in the evidence that appeals to our own senses, so the patient is apt to lay the more stress upon his ***subjective symptoms,*** while the physician is compelled to rely mainly on the objective ones in making his diagnosis. Probably subjective symptoms would be of equal, and sometimes of greater value than the objective if the power of conveying to the mind of another an accurate impression of our own sensations had not been denied us. We are able to locate more or less definitely the seat of a pain, and to describe it roughly as burning, throbbing, dull, or sharp, terms which must presuppose the experience of similar sensations on the part of the listener if they are to be understood, but the finer distinctions of quality, which often are apparent to the sufferer and probably would be of great service to the diagnostician if he could appreciate them, we are as unable to express as we are to convey the sensation of red to a person who is color blind. For this reason we have to regard subjective symptoms as less reliable than objective ones, but they must not be neglected. The evidence we gain from them is apt to be unsatisfactory, and may even be misleading in some cases, because of our own limitations in understanding the sensations of the patient, but, if we are to arrive at a correct diagnosis, it must be taken into account, along with that derived from the objective symptoms and from the history.

The ***objective symptoms*** are learned through our own observation, and it is essential that we should cultivate this faculty to the utmost. The sense of sight is called upon most often, which we must train to see the minutest details, but the sense of touch must be trained equally well to detect slight variations from normal in the tension. With the naked eye we make an inspection of the face, the lids, and all visible portions of the eyeball, and this usually suffices to reveal an external lesion, or to establish the fact that all of the visible tissues are in a normal condition, but in many cases we need to see more clearly by increasing the amount of light thrown upon the part in question, and perhaps by magnifying the image, before we can speak positively of its condition.

OBLIQUE OR FOCAL ILLUMINATION

Sometimes a surgeon may be seen to secure additional illumination by bringing a strong light near the eye, or by reflecting such a light into the eye by means of a head mirror, but such a method is very dazzling and painful to the patient, especially when he has a lesion which induces photophobia, like a foreign body on the cornea, and it is remarkable that anyone should continue to use such a crude means when a much more accurate and satisfactory light can be obtained easily and without irritation to the patient by oblique illumination. We seat the patient in a darkened room with a light two or three feet away, in front of and a little to one side of his face, and focus the rays of this light upon the tissue we wish to examine with a + 20 diopter lens, when all the details in the small illuminated area stand out very distinctly, not only because of the concentration of the rays of light, but also because the area of light is surrounded by a zone of darkness, and the contrast so afforded brings into relief slight disturbances that we would not be likely to see under any other conditions. If we need still more assistance we use a magnifying glass, or better a binocular loupe, in which magnifying glasses before the two eyes are adjusted so as to permit of stereoscopic vision of the enlarged image. In this way we can examine all parts of the cornea, anterior chamber, iris, and lens that are visible, and even the anterior portion of the vitreous without causing the patient any annoyance from the light.

OPHTHALMOSCOPY

Ophthalmoscopy is simply the inspection of the interior of the eye through an ophthalmoscope; so to complete the subject of inspection the method will be described here, although descriptions of other methods and instruments of observation will be found in connection with the conditions in which they are most apt to be used. The ophthalmoscope is simply an instrument that enables us to receive on our retinæ rays of light that are reflected from the funduses of our patients' eyes. There are many patterns of the instrument, but the principle is the same in all, that the rays of light shall seem to pass from our own eye into the eye of the patient, so that they shall return into our own, in accordance with a law of physics. This enables us to inspect such parts of the fundus as are not concealed behind other

tissues. *It is essential that the pupil should be dilated* if we are to make such an inspection thoroughly because it is likely to contract as a reflex response to the entrance of the light, especially when the latter falls near or upon the macula. Even though an expert may be able to get a fairly good view through a pupil of moderate size, he is often obliged to use a mydriatic, and no one who is less experienced should expect to be able to dispense with such a drug. A drop of euphthalmine, cocaine, or homatropine solution should be instilled into the conjunctival sac half an hour before the examination and repeated in ten or fifteen minutes. If cocaine is employed in both eyes at the same time care must be exercised to see that the corneæ do not become dry, as the result of an arrest of the act of winking, for the dry epithelium will interfere with the view, and also may inaugurate a keratitis. The patient and the surgeon should be seated beside each other facing in opposite directions, so that by inclining his head a little to one side the surgeon can bring one of his eyes directly in front of the corresponding one of the patient at a distance of eighteen to twenty inches. The surgeon holds the ophthalmoscope in his right hand and uses his right eye during the examination of a right eye, and uses his left hand and eye in the same way when looking into a left eye.

The first thing a beginner has to learn is how to manipulate the ophthalmoscope so as to throw light continuously into the pupil while he moves his own head backward, forward, to the right and to the left. The ability to do this comes with practice only, but is acquired more easily with an electric ophthalmoscope, in which the source of light is in the instrument itself, than with one by which a light at the side of and slightly behind the face of the patient is reflected into the eye, because the angle of incidence with which the rays fall upon the mirror changes with every movement of the observer, who consequently must keep the position of the mirror adjusted to every change that takes place. After a while he does this unconsciously. When the light is thrown into the eye from a distance of eighteen to twenty inches, and we look through the aperture of the instrument, we see a red or reddish glow in the pupil, which should be perfectly clear, and in which any opacities which there may be in the media stand out as black spots. This is the **red reflex of the fundus.** If we are looking directly at the papilla the color of this reflex is yellowish, or pink, but from all other parts of the fundus it should be a bright red. The tone of color varies; in blondes it usually is scarlet,

while in brunettes it more nearly approaches vermilion, though there are exceptions to this rule because the pigmentation of the fundus does not always correspond to that of the skin, and in negroes it is apt to be deeper—a brownish red. We have the patient turn his eye in various directions so as to be able to observe the reflex from all parts of the fundus, for any change in the appearance anywhere except at the papilla will indicate the presence of something wrong. Next we need to get a distinct view of the fundus, which we may obtain by either the indirect, or the direct method. Each of these has its own peculiar advantages and, as *Loring* says, "no one can be a good, or even a passable, ophthalmoscopist who cannot make use of both." The principal advantage of the indirect method is that as the image is magnified only about four diameters we are able to obtain a general survey over a rather large field, see the details of a considerable part of the fundus in their relations to one another, and locate pretty accurately the location of any lesion that may happen to be present. The advantage of the direct method is that as the image is magnified fourteen times we can study the details of a small area in the fundus much better, and can measure differences of level.

The Indirect Method

In the indirect method, which is known also as the **examination of the inverted image,** the patient is told to look past the ear of the observer at a distance of about four inches, or the surgeon may hold the handle of the ophthalmoscope inclined somewhat outward and extend his little finger still farther out for the patient to look past. This brings the papilla directly in front of the instrument. The surgeon then holds a + 13 diopter lens between the thumb and a finger of his disengaged hand 7 cm in front of the patient's eye, and focusses his own eye at a point 7 cm in front of the lens, where an inverted aerial image of a part of the fundus has been formed. He may do away with the effort to focus his eyes by interposing a + 3 diopter lens in the ophthalmoscope, and this is necessary when he is presbyopic, but it is better for a young man to gain the mastery over his own accommodation. The image is apt to appear indistinct at first, but is made clear by the surgeon moving his own head slowly back and forth, as one would adjust a microscope by means of the screw. If a distinct image cannot be obtained the reason may be because the surgeon, the patient, or both, has an uncorrected error of refraction, probably

astigmatism, because of dustlike opacities in the refractive media, or because of disease in the fundus; but faulty technique and refractive errors must be excluded before the indistinctness is ascribed to the presence of disease. If only a part of the papilla comes into view the surgeon moves his own head toward the side he wishes to see, or moves the lens to take advantage of its prismatic refraction. By having the patient move his eyes in various directions the entire fundus can be viewed to within 5 mm of the ciliary body. To see the macula mydriasis is necessary, and the patient is told to look directly into the aperture in the mirror. Reflexes of light sometimes bother even experts, who are accustomed to pay no attention to them. They come from the anterior surface of the cornea and from the surface of the lens which is used, which must be perfectly clean. Other sources of light than the one employed must be excluded, the lens may be moved or tilted slightly so that the reflexes will not be directly in the way, and then they must be ignored. Differences in level may be detected by parallactic displacement if we move the lens back and forth sideways a little during the examination, as the details that are nearer the observer seem to move more rapidly than those which are farther away. The top of a swollen papilla seems to move faster than the retina, and the margin of a glaucomatous cup seems to slide forward over the base.

The Direct Method

In the direct method, also called the **examination of the upright image,** the surgeon, as soon as he has obtained the red reflex, slowly approaches his eye to that of the patient, ever keeping the reflex in view, until the two eyes are close together, when a part of the fundus suddenly appears before him. During this time the patient looks straight ahead into the distance. The surgeon must keep his own accommodation relaxed, as though he were looking at a far distant object, but this is the only difficulty he experiences, aside from that of managing his light. If the image is not clear it is made distinct by the interposition of lenses in the ophthalmoscope. When the surgeon has his own accommodation under perfect control he can measure the hypermetropia and hypermetropic astigmatism of the patient's eye in the great majority of cases, by noting with what lenses the retinal vessels at the margin of the disk come most plainly into view in the different meridians of the

eye; but he cannot measure myopia with an equal accuracy. Differences of level in the fundus are measured in the same way, as a difference of refraction of three diopters corresponds to an elevation or a depression of 1 mm. When the difference of level is great we can bring out the details at various points by a successive change of the lenses and so get a mental picture of the whole, as when we follow the vessels from the retina to the top of a detachment by the interposition of stronger and stronger convex lenses.

HISTORY TAKING

The examination of the eye properly includes an inquiry not only into the present subjective and objective symptoms, but also into any past conditions of the patient, of his family, or of his ancestry, that may have a bearing on the ætiology of his trouble. This is called **taking the history.** All that is sought in any case is sufficient evidence to render the diagnosis unquestionable, so in some cases there is much that need not be inquired into, while in others we have to delve deeply into all of these matters. It would be ridiculous to inquire into the family history, unrelated subjects of his own past history, or to make an exhaustive examination of the eyes of every patient who chanced to have a cinder beneath his lid or in his cornea, yet we meet with conditions in the eye which are to be explained only through the effects produced by a former infection or disease, and others which are hereditary or familial. The importance of the history is conceded universally, yet the taking of it is greatly neglected. To many minds it seems to mean that we should sit down with pencil and paper, question the patient, beginning with his ancestry, write notes on all manner of unrelated subjects, and work down gradually to his present condition. Such a method is not only difficult and wasteful of time, but is asinine. It antagonizes the patient, who cannot see the bearing of such an interrogation, and therefore replies carelessly, if not impatiently, and this, instead of aiding us, makes our task more difficult. Nature has granted to some more than to others the power to draw out essential points from people of the most diverse characters and dispositions, but we need to cultivate such power as we have until it takes only a few moments to learn the facts needed for our guidance. A certain routine should be followed, but not the one

indicated above. The following may serve as an example, though it is no better than any other that is equally thorough.

While the patient is telling what he wishes to say we listen attentively, jot down some notes, perhaps, but rely mainly on memory for the present, and at the same time glance over the entire face, with especial attention to the regions of the preauricular gland, the lacrimal sac, the lids, and their margins. Without interrupting the narrative we press gently upon the lacrimal sac and observe if there is any regurgitation from the punctum, draw down the lower lid so as to reveal its palpebral conjunctiva, inspect it, and then in turn the bulbar conjunctiva, the caruncle, the semilunar fold, the cornea, the iris, and the anterior capsule of the lens, and take the tension of the eyes with the fingers, for this much of the examination is to be made in almost every case. By this time we have the principal subjective symptoms in mind and may be guided by them, or by objective symptoms we have discovered, to devote attention to some particular tissue first of all, perhaps to study it by oblique illumination. If no external lesion has become apparent we may evert the upper lid at this time, or defer the eversion until after the functional tests, which are pretty sure to interrupt the narrative, if it has not been completed. We first test the pupillary reactions, determine the vision of each eye separately, measure the corneal astigmatism with the ophthalmometer, ascertain the manifest refraction and the range of accommodation, investigate the balance of the extrinsic muscles, and then invite the patient into the dark room for an ophthalmoscopic examination. We should record all of our findings by these tests immediately. It now becomes necessary in many cases to dilate the pupils with a mydriatic, or to paralyze the ciliary muscle with a cycloplegic, and we have to wait until the drug has produced its effect before we can proceed. This is a good time to inquire into the history. Sometimes it is well to begin by rehearsing briefly our conceptions of the subjective symptoms and asking corrections; at other times this is unnecessary. If we start by trying to ascertain as nearly as possible the date of the onset of the affliction and the nature of the early symptoms, the patient is apt to follow our lead readily when we inquire into his past troubles, his occupation and habits, and finally into his family history, for he is led to believe that the questions have been called forth by what we have found, and to take an intelligent interest in answering them. Very little time is taken in doing this,

unless the answers lead us to further investigation along some particular line, while the patient, so far from being irritated, is likely to be impressed favorably by our interest in his case.

Inquiry into the family history is out of place when the trouble is a purely local one with which heredity is known to have nothing to do, but when we find a condition in which the ætiology is obscure, or one in the production of which heredity may have played a part, we should encourage the patient to rack his brains to furnish us as much information as possible about other members of his family, near relatives, and ancestry. When the same defect is present in several children of the same family, though not in the parents or near relatives, we need to inquire whether a grandparent, a great-grandparent, or any member of their families is known to have suffered in a similar way. A defect that reappears in widely separated generations, skipping the intermediate ones, is called atavistic. It is exceptional for us to be able to get a family history that covers more than three generations, and often we cannot get even this, so the results of this inquiry are apt to be incomplete and unsatisfactory. When a defect in several members of a family cannot be traced back, and is not known through other researches to be atavistic, we call it familial. Other hereditary defects are transmitted from one generation to the next, perhaps directly from parent to child, but often indirectly from an uncle or an aunt to the younger victim. Occasionally they seem to be transmitted through unaffected females to the males of the family. Some hereditary troubles are not apparent at birth, but develop in the younger person at about the same age that they did in the older one, and in such cases the family history throws light not only on the diagnosis, but also on the prognosis.

In a comparatively small, though numerically large, number of cases we still need a report from the pathologist on a bit of tissue that has been excised for examination, on a culture taken from some discharge, or on the blood and excretions of the body, before we shall have amassed all of the symptoms obtainable.

DIFFERENTIATION

Even though each symptom alone is of little value, we shall find that when a number are taken together, some one, or some small group stands out prominently as indicating the tissue that is the

site of the lesion, or a definite fault of function. The other symptoms enable us to place the trouble in a general group which includes many that are quite diverse, and then the presence or absence of certain characteristic symptoms empowers us to exclude one trouble after another until perhaps only a single one is left. Then we have made a diagnosis by exclusion. The differentiating characteristics may be subjective, objective, or contained in the pathologist's report. The differentiation of eyestrain due to a refractive error depends on the relief of the subjective symptoms by the correction, that of an iritis from a glaucoma on the objective signs, that of a diplococcal from a pneumococcal ulcer of the cornea in an elderly person on the bacteriological findings. A correct diagnosis is imperative in each of these four pathological conditions, for each disease will ruin the eye if it is neglected, and the right treatment for each will aggravate and make worse the one from which it needs to be differentiated. Every lesion in the human body may be supposed to interfere with certain tissues and functions in a definite way, and probably we could make the diagnosis rightly in every case if we could be sure that we appreciated every symptom presented at its true value, but as we cannot do this we make mistakes. When two persons suffer from the same trouble the symptoms will not be duplicated exactly unless the lesion is exactly the same in position, extent, and every other characteristic in both, so the clinical pictures must vary more or less. The characteristic symptoms are not as pronounced in some cases as in others, and sometimes those presented are actually misleading, as in the early stage of some cases of iritis, when the redness of the eyeball may be fairly uniform, the pupil slightly dilated, and the conjunctiva have a little secretion, circumstances under which many an expert has been deceived. Finally, the symptoms indicative of diseases of the eye are so interwoven in some cases with those produced by the diseases of the general organism that they can scarcely be disentangled, and many lesions of the eye are symptomatic of diseases of other organs of the body, so occasionally we have to make an excursion into the domain of general medicine, and call in the aid of other practitioners, in order to make a correct diagnosis.

CHAPTER II

The Lids

When we begin an examination of the eyes we note first the relative positions of the orbits, any swelling of the face, or other morbid condition, especially on the side of the affected eye, and note any swelling in the region of the preauricular gland, or of the lymphatic glands of the neck. Then we note the position of each lid, the condition of its skin and of its margin, the presence of any liquid or dry discharge between or on them, and of any scurf, scabs, sores, swellings, or other abnormality that may be present. Sometimes we need to observe the movements of the lids in the act of winking, or to test their sensibility when there is any reason to think of a possible anæsthesia or hyperæsthesia.

THE LIDS

Certain defects may be present in the lids, and we need to know whether they are congenital or acquired. A lid that is too short, partially or wholly absent, may be congenital, but in that case cicatrices are absent, and there is no history of traumatism. A **coloboma** may vary in size from a notch at the margin, to a fissure which runs the whole width of the lid and involves its entire thickness. If it is artificial we find its margins cicatricial and can obtain a history of traumatism, as a rule, while if it is congenital its margins are covered by conjunctiva, sometimes are attached to the surface of the eyeball by synechiæ, or the defect is associated with dermoid, and the condition has existed since birth. Very rarely two colobomata may be present with a semi-detached piece of the eyelid between them, resembling a double harelip.

EPICANTHUS

The base of a baby's nose frequently appears to be too broad, with each of the inner canthi covered more or less by a crescentic fold of skin. This condition persists normally among the Mongo-

lian races, but among Caucasians it usually disappears in a few years with the growth of the child. Occasionally it persists and then forms a congenital anomaly known as epicanthus, which almost always is bilateral and not to be mistaken for anything else. In the majority of cases it is slight, and of importance only in so far as it impairs the beauty of the face, but sometimes the folds of skin are so large as to cover not only the canthi and the caruncles, but also a large part of the inner portions of the lids, giving rise to a serious deformity and interfering with vision. These pronounced cases are apt to be associated with other congenital anomalies, especially ptosis, and strabismus may be simulated even when it is not present.

PTOSIS

One or both of the upper lids may droop; this is called ptosis. This condition varies in degree from a very slight droop that gives the face a sleepy expression, to one in which the lid hangs down loosely and covers the pupil, rendering the eye practically blind. It may be congenital or acquired. The differentiation is made usually from the history and the presence or absence of lesions known to be capable of its production.

Congenital Ptosis

The testimony of the parents that the droop has been present since birth, or of the patient himself that he has had it as long as he can remember, ordinarily suffices for a diagnosis of congenital ptosis. The only probable chance of error is in the case of the rare disease chronic progressive ophthalmoplegia, but this can be excluded by an examination into the motility of the extrinsic muscles, for although this may be impaired to a greater or less degree in both, secondary contractures will be found in the muscles only when this disease is present. If the ptosis is associated with epicanthus, strabismus, or nystagmus, or if there is no diplopia when the lid is lifted, we may be pretty sure that the ptosis is congenital. The same is true if we find it accompanied by microphthalmos, coloboma of the uvea, ectopia, or congenital cataract, or by malformations in other parts of the body, or by mental inferiority. When the family history can be obtained we may be able to trace the defect through several generations, though frequently we meet with isolated cases. We are strongly impressed with the probability that the defect is

hereditary if the lid exhibits anomalous movements, as when it is lifted during chewing or swallowing.

The **picture presented by cases of congenital ptosis** in which the pupils are partly covered is characteristic. The wrinkles into which the skin of the lid is thrown by the action of the levator are smoothed out, the skin of the forehead presents arched wrinkles caused by the efforts of the occipitofrontalis to lift the lid, which also draw the eyebrows upward and outward so as to give the face a peculiar expression, while the patient carries his head tilted backward so as to avail himself as much as possible of his narrow palpebral fissure. The facial appearance, the smooth lids, and the backward tilt of the head form the syndrome of Hutchinson.

The cause of the ptosis is a paresis of the levator due to a fault located in the muscle itself, in its nerve center, or in the connecting neurons. The muscle may be absent, poorly developed, or have its fibers of attachment erroneously distributed and inserted; the nerve center may be absent or only partly developed, the conducting neurons may be absent or imperfect; but these causes rarely can be differentiated. The degree of paresis cannot always be determined from the extent to which the lid droops, for if the skin of the lid is long and lax, the contraction of an incompletely paretic levator may not suffice to lift it, while if it is short a total paralysis may seem to be partial, especially when the action of the occipitofrontalis is very effective. The simplest way in which to estimate the degree of the paresis is to press upon the margin of the orbit with the finger so as to fix the skin and exclude the action of the latter muscle, and then to note the effect produced when the patient tries to open the eye.

Acquired Ptosis

Acquired ptosis is symptomatic of a central or peripheral nerve lesion, and must be considered not as a condition to be dealt with, but as a guide to the lesion. It may be the result of certain conditions in the lid itself, in the orbit, in the accessory sinuses, in the central or sympathetic nervous system, of traumatism, of poisoning, or of some general disease. Any change that increases the weight of the lid to a point at which it overbalances the strength of the levator will cause it to droop to a degree proportionate to the overbalance. This **mechanical** ptosis may be caused by tumors, inflammation and œdema of the lid, hypertrophic and redundant skin,

trachoma, certain skin diseases like elephantiasis, and the binding down of a lid by a symblepharon. Such causes are quite evident as a rule, but it may be well to define at this point what we mean by **pseudoptosis.** Some authors apply this term to any droop of the lid which is not dependent on a paresis of the levator; others to one due to an abnormal weakness of the levator; still others to one produced by spasm of the palpebral fibers of the orbicularis. The line between it and true ptosis is hard to draw in both of the first two definitions, while it is well marked in the third, so we shall consider pseudoptosis under blepharospasm.

Sometimes we meet with a **senile** ptosis in aged people. As age advances all of the muscles, including the levator, grow feeble, the skin becomes withered and wrinkled, the orbital fat atrophies and allows the eyeball to sink back so as to give the lid less support, while the weight of the latter increases, and senile ptosis is the result partly of the recession of the eyeball, partly of the weakness of the muscle, partly of the greater weight of the lid produced by changes which may not be of the same nature in all cases. Whether *Fuchs* is right when he says that the fibrous bands which attach the levator to the skin and the upper margin of the orbit become stretched until they no longer support the weight of the lid, or *Lodato* is correct in thinking that the blood vessels become enlarged with the atrophy of the skin and elastic fibers, or both are right in some cases and wrong in others, the syndrome of senile ptosis consists of old age, a withered, wrinkled, frequently yellowish skin that hangs in pouches, deeply set eyeballs, and drooping upper lids. This ptosis always is bilateral.

An isolated bilateral ptosis, which seems to be a variety of the senile and is met with almost wholly in old women, is a rare condition which was described first by *Fuchs.* It becomes more marked when the patient is tired, and it is at such times that attention is likely to be called to it by a corrugation of the forehead caused by the effort of the occipitofrontalis to lift the lid. We do not know yet whether the cause of this is a nuclear affection, or a primary atrophy of the levator.

Traumatism may induce ptosis by a local injury to the levator itself, to its insertion into the tarsus, or to its nerve. The oculomotor nerve may be wounded or compressed within the orbit, or within the skull, or the injury may be located in its cortical, or its nuclear center. The history, associated perhaps with a local swelling and

discoloration, may indicate a contusion to be the cause. A shot or stab wound properly situated may make it almost certain that the muscle or its attachments have been divided. A scar in the brow is not necessarily connected with a ptosis, but sometimes it will prove an excellent guide, as in a case in which such a scar attracted my attention and led to the drawing out of a history of a severe contused and lacerated wound received from a blow with a beer bottle during a drunken brawl. The patient stated that the swelling of the tissues lasted a considerable length of time, and he could not remember that he had opened the eye since he received the blow. A large piece of the bottle was found near the apex of the orbit, whence it was removed with some difficulty; healing followed with practically complete restoration of its function to the levator.

This muscle may be affected in the same way by the **pressure of a hemorrhage** coming from a fracture of the wall of the orbit, or the laceration of an orbital vessel, when we shall find more or less protrusion of the eyeball, impaired motility of the extrinsic muscles, and a suffusion that appears first beneath the bulbar conjunctiva, later beneath the skin of the lids.

The ptosis occasionally seen in infants after delivery with forceps usually is due to some lesion that has been produced in the orbit, but sometimes indicates an injury to the oculomotor nerve, or to its cortical or nuclear center.

A ptosis that appears after a **fracture of the base of the skull** indicates that the oculomotor nerve has been injured at some point in its course. When it is associated with a pulsating exophthalmos and a paralysis of the other muscles supplied by the oculomotor, we know that the nerve has been wounded along with the carotid at the cavernous sinus. Ptosis combined with paralysis of the external rectus shows that both the oculomotor and the abducens have been torn or compressed along the petrous portion of the temporal bone. A ptosis that becomes apparent gradually after a fracture of the base, along with a dilatation of the pupils, a paralysis of the hypoglossus, and a loss of the pharyngeal reflexes, may call attention to the development of a fatal purulent meningitis.

Foci of softening frequently appear in the brain after injuries to the skull, and when such a focus involves the cortical or nuclear center of the oculomotor nerve ptosis will be produced, usually with

paresis of other nerves whose centers are adjacent and involved. It is an important fact to remember that such lesions may develop a long time after an apparent recovery from an injury to the skull, so the subsequent appearance of a ptosis may be a very significant symptom. If it is accompanied by glycosuria the focus of softening is located in the nuclear center.

A combination of ophthalmoplegia, of which ptosis forms a part, associated with stupor, delirium, and sometimes optic neuritis or partial optic atrophy, occurs frequently in ptomaine poisoning and chronic alcoholism, less often in poisoning with lead and carbon dioxide, as well as in uræmia, and indicates that the condition of the patient is very grave.

Ptosis occurs quite rarely in the course of an acute infectious disease, though it has been known to appear in epidemic cerebrospinal meningitis, influenza, typhoid fever, measles, pneumonia, and rheumatism. Even after diphtheria it occurs in less than one per cent. of the cases of postdiphtheritic muscular paralysis.

Syphilis often causes ptosis, either by means of a tarsitis that increases the weight of the lid; a gummatous periostitis of the margin of the orbit, which acts partly through the thickening produced, partly through pressure; a mass in the orbit that produces exophthalmos with restricted movements of the eyeball; a periostitis about the superior orbital fissure that affects the oculomotorius, the trochlearis, the abducens, and the upper branch of the trigeminus, when a complete ptosis will appear suddenly together with a paresis of all the muscles of the eye, and an anæsthesia of the cornea, conjunctiva, and the skin supplied by the first branch of the trigeminus. Any of these combinations of clinical symptoms is suggestive of syphilis and should lead immediately to a thorough investigation of the organism for other corroborative indications. But these are not the only syndromes of syphilitic origin in which ptosis forms a part; the lesion may be within the skull. Associated with paresis of other cranial nerves and such general cerebral symptoms as weakness of memory, or disturbances of consciousness, it is apt to point to a gummatous neuritis or perineuritis somewhere along the course of the motoroculi, generally associated with a basal meningitis. The usual location of a gummatous basal meningitis is in the deep recess between the chiasm, the peduncles, and the trunks of the oculomotor nerves, and in such cases the ptosis is bilateral. A gummatous lesion at any point along the course of

the nerve, from and including the nucleus, may produce a ptosis without affecting any other of the muscles which it supplies, and then the differentiation has to be made from such localizing symptoms in other parts of the body as may be produced by the lesion on other nerves. A gumma, or a focus of softening in the peduncle may affect the root of the nerve, and when we meet with an isolated ptosis together with a hemiplegia of the opposite side of the body, we believe that the lesion is in either the pons or the peduncle. The region of the nucleus of the oculomotor nerve may have its nutrition impaired by disease in the basilar artery, may be the seat of a gumma, or may undergo primary atrophy during the virulent stage of syphilis, and a syphilitic nuclear lesion may be diagnosed when the external muscles of the eye supplied by this nerve become paretic one after another while the internal ones remain intact, the pareses are bilateral, sugar appears in the urine, and the disease has remissions. A nuclear lesion is excluded by paresis of one or of both sides of the body, optic neuritis, and optic atrophy. Temporal hemianopsia shows that the chiasm is involved, homonymous hemianopsia that one tract is affected back of the chiasm. Syphilis does not produce a supranuclear ptosis.

Ptosis is a symptom in many **diseases of the central nervous system.** It is rare in general paresis, in which it is found in only two or three per cent. of the cases not complicated by tabes. In tabes dorsalis, or locomotor ataxia, on the contrary, it is common, and often is one of the initial symptoms of the disease. A very marked peculiarity when it appears in the early stage is that it is apt to be of a transient nature, so that a ptosis of one or both eyes which disappears in a short time without treatment, or is recurrent, should lead us at once to look for other symptoms, but in other cases we may find it associated with a paresis of the abducens, or to be the first sign of an advancing external ophthalmoplegia. Another disease in which the onset of ptosis is an initial symptom in about ten per cent. of the cases is multiple sclerosis; in this disease it affords material aid to the diagnosis, which often is very difficult in its early stages. Associated with weakness of the muscles of mastication and of the throat ptosis indicates asthenic bulbar paralysis. It forms a part of the chronic ophthalmoplegias met with in diseases of the medulla oblongata and of the prosencephalon. A curious form of chronic progressive ophthalmoplegia, which is rare and of unknown cause, begins with a ptosis in early

childhood and advances slowly to become total at the end of years; the ptosis in this disease may be mistaken for the congenital form unless we note the presence of contractures in some of the muscles.

Occasionally ptosis occurs in connection with such intracranial lesions as hemorrhages, tumors, abscesses, and embolic or thrombotic softenings of the brain, and then may serve as a localizing symptom, as it shows an involvement of some part of the oculomotor tract, either along the base of the brain, in the peduncles, in the tegmentum, in the thalamus, or in the nuclear region; in which of these places the lesion is situated must be determined from other symptoms. Associated with bitemporal hemianopsia ptosis is a symptom of hypophyseal disease. A tumor of the cerebellum can produce ptosis only when it presses on the corpora quadrigemina. Ptosis of one eye associated with paresis of the facial and hypoglossal nerves on the other side indicates a lesion to be situated in the peduncle. A bilateral ptosis without hemianopsia, but with other cerebral symptoms, points to the region of the nucleus as the situation.

Sometimes the development of a ptosis is useful in the differentiation of the various forms of **meningitis;** it is rarely met with in the epidemic cerebrospinal variety, occurs in the purulent only when the inflammation is situated at the base of the brain, and when it develops slowly we may be pretty certain that the patient is suffering from the tubercular form.

Other diseases in which ptosis is met with are Graves's disease or exophthalmic goiter, acute poliomyelitis, multiple neuritis, Landry's disease, Gerlier's disease, herpes zoster, ophthalmic migraine, and polymyositis. A moderate ptosis of one eye, associated with a reduced tension of the eyeball, a slight enophthalmos, and myosis, suggests that some lesion involves or presses upon the ganglia of the **cervical sympathetic nerve** on the same side; the lesion may be a swollen cervical gland, a goiter, a neoplasm of the œsophagus, an intrathoracic growth, an aneurysm of the aorta, an abscess of the mediastinum, or an operative removal of the superior cervical ganglion. At first we find also a hyperæmia of the conjunctiva, a flushing of that side of the face with anæsthesia, a local elevation of temperature and increased secretion, but later the skin of that side of the face atrophies and becomes pale, the local temperature is lowered, and the secretions are diminished. This form of ptosis is unilateral almost invariably, and is due to a paralysis of Mueller's

muscle. A ptosis of this nature is met with occasionally in syringomyelia.

Finally, ptosis may be caused by **hysteria,** and we suspect this to be the cause when the lid falls more quickly after it has been raised by the finger than is usual in the paralytic form. In many cases hysterical pressure spots can be found, where pressure with the finger will cause the eyes to open instantly. These spots are likely to be found in the same places as in hysterical blepharospasm.

WINKING

The act of winking is partly voluntary, partly reflex. Ordinary winking is an involuntary reflex excited by stimulation of the filaments of the trigeminus through a slight dryness of the surface of the cornea. The levator is relaxed while the palpebral fibers of the orbicularis contract so as to move the upper lid downward, and both lids slightly toward the nose, a movement which spreads a film of moisture over the surface of the eyeball, removes any dust that may have fallen upon it, and propels the tears into the lacrimal lake. The frequency of the act is given by some authors as from two to four winks per minute, by others as from five to ten, but it varies a great deal within normal limits, and is affected by various conditions, such as local or general fatigue, so it must be increased or decreased considerably in order to become a noticeable symptom.

Abnormally infrequent winking is one of the symptoms of exophthalmic goiter, or Graves's disease, but commonly it indicates that the surface of the cornea is insensitive, whether made so by the instillation of an anæsthetic like cocaine, or by a lesion of the trigeminus. When the corneal anæsthesia of both eyes is profound the act of winking is nearly or quite abolished, the epithelium of the cornea tends to become dry, and a keratitis may be started which will develop into a keratitis e lagophthalmo if the anæsthesia persists long and the eyes are not protected from desiccation. Very slow winking, in the absence of a local anæsthetic, should lead us to search for a lesion that inhibits the function of the ophthalmic branch of the trigeminus.

The lids respond by the same act to protect the eye when it seems to be threatened by an approaching body, and this reflex may be utilized sometimes to determine whether a suspected hemianopsia is present or not; if we move an object rapidly toward the

eye in the field that is supposed to be blind and the lids close, we know that the eye perceived the movement, but if they do not move the object was not seen. Voluntary winking is used mainly as a test for paresis of the orbicularis; if the patient cannot close the eye perfectly this muscle has lost more or less of its function.

Many healthy people exhibit a peculiar vibratory **tremor** of the lids when they keep their eyes gently closed, but such a tremor is more distinct in neurasthenia. Some have fibrillary twitchings of individual bundles of fibers of the orbicularis which not only can be seen by others, but are felt by the patient; when these are transient they are apt to be the result of either sexual excesses, great fatigue, or hard, near work of the eyes in persons who are anæmic or neurasthenic, but when they are constant they form what is known as muscular tremor, which is met with in cases of facial paresis and of migraine.

BLEPHAROSPASM

Blepharospasm is an abnormal closure of the lids produced by a spasmodic action of the orbicularis. It appears in two forms, clonic and tonic. In the clonic form the spasm is of short duration and soon recurs, in the tonic the lids remain closed for some time. A **clonic** spasm may be described as an accentuated form of winking, the act being more forceful, more frequent, and more prolonged than normal. We see it frequently in children who have some slight irritation of the eyes, such as may be produced by an error of refraction, or a follicular conjunctivitis. When such a nictitation persists after the local irritation has been removed the child usually is anæmic and nervous, and in need of a general tonic treatment. Clonic spasms are met with also in chorea, and in what is called habit chorea, when they are associated with twitchings of the mouth and shakings of the head. Sometimes a single clonic spasm is produced when a person goes suddenly from the dark into a bright light, and then it is apt to be preceded by a sneeze.

A **tonic** blepharospasm commonly indicates trouble on the surface of the eye, the presence of a small foreign body, the scraping of eyelashes, a phlyctenular keratitis, an interstitial keratitis, or an erosion of the corneal epithelium by some other cause. It is met with occasionally in nearly all the inflammations of the conjunctiva, cornea, and iris. Sometimes it occurs in trigeminal neuralgia, in herpes zoster, and in diseases of the nose, mouth, pharynx, and

teeth that irritate other branches of the trigeminal nerve. Usually the cause is easy to detect in all of these cases, except the last class, in which it may be recognized only after its removal, as when a persistent blepharospasm disappears immediately after the extraction of a carious tooth.

Blepharospasm may be caused also by anything that irritates the facial nerve at any point in its course from its nucleus to its peripheral filaments. It forms a part of the facial spasms that are excited by an irritative lesion at the nucleus of the nerve; it appears together with spasms and pareses of muscles on one side of the body when the cortical center is irritated, and therefore is met with among the symptoms of Jacksonian epilepsy; it may be excited by tumors and aneurysms at the base of the brain, by meningitis, by troubles in the ear and temporal bone, and by scars on the forehead or cheek. In all of these cases the cause is to be learned through the accompanying symptoms.

A strong light is rather apt to excite a blepharospasm in nervous or hysterical persons, and sometimes does so in others when a dazzling is produced by opacities in the refractive media, such as occasionally happens in incipient cataract. An occupational blepharospasm is met with in watchmakers who have long been in the habit of holding a magnifying glass in the grip of the orbicularis.

In many cases we have difficulty in explaining the occurrence of a blepharospasm when it is a symptom of a traumatic neurosis that follows a slight injury to the head, or appears during an attack of migraine, or in hysteria, as in these cases it does not seem to be dependent on any observable lesion. When such an unexplainable blepharospasm is associated with a contraction of the visual field, an inversion of the color fields, monocular diplopia, photophobia, contractures of the muscles, or anæsthesia of the skin of the lids, all unexplainable by any determinable lesion, it may be pronounced hysterical. Such a blepharospasm usually is bilateral, but it may affect only one eye, and then if the palpebral fibers of the muscle alone are involved, it may simulate ptosis by producing the condition known as pseudoptosis. In some cases it may be arrested by pressure on certain tender points, which usually are situated along the course of the trigeminus, as at the infraorbital and supraorbital foramina, but often are found elsewhere. *Roemer* tells of a patient whose blepharospasm was stopped by pressure on the tip of the nose, while other observers have found such points

in the nose, in the mouth, on the cartilages of the ribs, and on the vertebræ. Conversely, pressure on various parts of the body has been known to excite a blepharospasm in hysterical people.

Tonic spasm of the palpebral fibers of the orbicularis is able to keep an eye closed for a long period of time and to simulate a unilateral ptosis. This is **pseudoptosis,** and is hysterical in most cases. The differentiation from true ptosis is made from the facts that the forehead is not corrugated, the brows are low and straight, and the skin of the lid is not smooth and lax. Sometimes the eye can be observed to open in a moment of excitement.

Some old people suffer from a **senile** blepharospasm that recurs at rather short intervals, comes on suddenly and keeps the eyes closed for quite a length of time. At the moment of attack the margin of the lower lid may be seen to rise, that of the upper one to sink, and both to move toward the median line of the face, while curved transverse folds form in the skin of the lid and about the eyes, and the patient distorts his face and opens his mouth in an effort to overcome the spasm. Attacks of this nature have placed old persons in great danger while walking on the street, as they were suddenly rendered practically blind. The cause of this trouble is unknown.

ŒDEMA OF THE LIDS

The skin of the eyelids is very thin, and is attached so loosely to the subjacent muscles that collections of serous fluid frequently form beneath it and distend the subcutaneous tissue. This œdema can be recognized easily from the fact that the swollen lid pits on pressure, while the finger feels no crepitation, and the swelling often can be reduced considerably when the pressure is maintained. It may or may not be inflammatory, may affect the upper or the lower lid, or both, and may be unilateral or bilateral. The skin is reddened in the inflammatory variety, paler than normal in the other. Œdema is symptomatic of so many local and general lesions that all cannot be enumerated, but a few will be mentioned to try to impress the fact that its presence is an urgent indication for a thorough and exhaustive inquiry into the condition of the organism until its cause shall have been ascertained.

A **chronic, noninflammatory, bilateral œdema,** usually of the lower lids, may be due to heart disease, nephritis, a hydræmic con-

dition of the blood, chronic arsenical poisoning, or trichinosis. A similar œdema of shorter standing in the lids of either one eye or both may indicate an earlier stage of any of the above conditions, or an inflammation in one of the accessory sinuses. An œdema that lasts a few days, with no inflammation or itching, and then disappears to recur in a few weeks, may indicate the onset of blepharochalasis, but is also suggestive of Quincke's disease, in which it may appear alone or in association with a similar œdema of the skin elsewhere, or of the mucous membranes. This disease is not yet fully understood, but it is thought to be an angioneurosis of central origin. Another disease in which œdema is a symptom is myxœdema, in which the swollen lids protrude and the skin is pale and cool.

An **inflammatory** œdema may be due to traumatism, as in a black eye; to a focus of inflammation in the lid itself, as in a hordeolum, or the sting of an insect; to inflammation of neighboring tissues, as in dacryocystitis or periostitis of the margin of the orbit; to acute inflammation of the eye, such as conjunctivitis, iritis, glaucoma, and panophthalmitis; to an inflammation in the orbit; or to an inflammation in one or more of the accessory sinuses. Hence, a careful examination must be made of the eye itself and of its appendages in every case of œdema of the lids. When the lower lid is more swollen than the upper, perhaps feeling brawny while the upper one is soft, and the swelling extends down along the side of the nose, we have reason to suspect an acute dacryocystitis, while if the region along the nose is approximately normal and the middle of the swelling is directly beneath the eye, the focus of inflammation is more likely to be in the lower part of the orbit when it cannot be found in the lid itself. A sudden inflammatory œdema of the upper lid that is not associated with an inflammation of the eye, and is not explained by traumatism, or a local focus of inflammation, leads us to suspect inflammation in one of the accessory sinuses, and we feel particularly confident of this diagnosis if the swelling is mainly in the inner third. When the outer part of the upper lid is chiefly affected we think rather of a possible abscess, an orbital cellulitis, or an acute dacryoadenitis. When no cause can be detected we must remember that an œdema of the lids may be a forerunner of a trouble that has not yet made itself manifest. *Beard* states that an alveolar abscess has caused a palpebral œdema before it was known that there was any dental trouble. A recurrent or fugitive œdema associated with

pain, replaced at times by a blackening suggestive of ecchymosis, sometimes is indicative of sinus disease.

The so-called **solid œdema** is a condition in which the lids are enlarged enormously with no signs of inflammation or of involvement of other parts. *Ball* describes the swelling as soft, elastic, pitting on pressure, and of a dusky reddish brown color, and states that in the majority of cases it has followed attacks of erysipelas, as well as that some cases have terminated in tuberculosis of the conjunctiva. The affection seems to be due to an obstruction of the lymphatic circulation, but its nature is not clear.

Malignant œdema may appear as a sequel to, or as a precursor of an attack of anthrax. The color of the skin is pale at first, while the lid has a doughy consistence, but later it becomes cyanotic, bullæ appear, and patches of necrosis are formed. The diagnosis is established by the other symptoms and the demonstration of anthrax bacilli.

EMPHYSEMA OF THE LIDS

When we press our fingers upon what appears to be a noninflammatory œdema of the lids and feel crepitation, we know that the subcutaneous tissue is filled with air instead of fluid, a condition called emphysema. The air has entered through a fracture or other opening in one of the bones of the orbit which is so situated as to open communication between the subcutaneous tissue and the cavity of the nose, or one of the accessory sinuses.

ECCHYMOSES OF THE LIDS

Hemorrhages into and beneath the skin of the lid are produced very commonly by contusions, penetrating wounds, and operations in which the free escape of the blood is impeded, when the history and the associated œdema or wound clears up the diagnosis. A trivial injury may cause a large ecchymosis in hæmophilia. Similar hemorrhages occur in children during paroxysms of whooping cough, and in elderly people, whose blood vessels are brittle, as the result of violent efforts, vomiting, or straining at stool. They may be produced also by severe compressions of the thorax and abdomen, and often are symptomatic of fracture of the bones of the orbit, or of the base of the skull. In these cases there may be little or no œdema, though this is not always so, and the absence of œdema goes to prove that the discoloration was not caused by a direct traumatism.

When it is due to a fracture of the base the subdermal ecchymosis is apt to be located near the inner canthus, usually is preceded by a hemorrhage beneath the conjunctiva, and may appear shortly after the accident, or not for several days. Acute exophthalmos with subconjunctival and subdermal ecchymoses shows that a large hemorrhage has occurred in the orbit. A fugitive blackening of the lids that resembles ecchymosis and recurs with attacks of pain, with an absence of discomfort in the intervals, should direct our attention to the accessory sinuses.

Minute hemorrhages scattered about in the skin are called **petechiæ.** Whether they are found in the lid or elsewhere they are apt to be symptomatic of such diseases as the hemorrhagic diathesis, purpura hemorrhagica, scurvy, and sepsis.

HYPERÆMIA OF THE SKIN OF THE LID

An ***active*** arterial hyperæmia characteristically accompanies an inflammatory œdema of the lids. A ***passive*** arterial hyperæmia may be produced by the presence of an aneurysm in the vicinity. A bluish red cyanosis with dilated, tortuous veins, a venous hyperæmia, signifies that there is an obstruction to the venous circulation, which may be general or local. It is met with when the foramen ovale of the heart is open congenitally, when the pulmonary artery is stenosed, when the flow through the jugular vein is impeded, and in such local troubles as thrombosis of the orbital veins, compression of the palpebral veins by retrobulbar tumors, and exophthalmic goiter. Both arteries and veins are dilated when there is an arteriovenous aneurysm of the internal carotid and the cavernous sinus, a condition that is further characterized by a pulsating exophthalmos.

ERUPTIONS AND DISCOLORATIONS ON THE LIDS

The eruptions of the acute infectious diseases, and of the cutaneous diseases which appear on the face, are to be seen on the lids, but most of them present nothing special in that location. Chicken pox seems to have a preference for the skin of the lids, and *Oliver* has ascribed urticaria of the lids to eyestrain in some cases. Usually the lids are spared in eczema of the face, but occasionally they are involved, and repeated attacks of this disease may cause the skin to be thickened permanently. Eczema is looked upon by many as the

fundamental trouble in phlyctenular conjunctivitis and keratitis, in which fissures are apt to be formed in the skin, especially about the outer canthus, and to occasion blepharospasm. The lids often are involved in facial erysipelas, which may cause abscess or gangrene, and repeated attacks are apt to result in a permanent thickening of the skin, or the condition known as solid œdema.

Herpes Zoster

An eruption of vesicles on the upper lid, the forehead up to the edge of the hair, and on the nose, sharply delimited by the median line of the face, accompanied by severe neuralgic pain, fever, and prostration, is characteristic of herpes zoster. This disease is most common in elderly and feeble patients, but is not rare in the young and well nourished. The ophthalmic branch of the trigeminus is the one affected when the above picture is presented; when the second branch of the same nerve is attacked the eruption appears on the lower lid and over the superior maxillary and malar bones, likewise sharply demarked by the median line of the face, a limitation that is present almost invariably. The eruption begins with groups of papules that vary in number and quickly change to vesicles ranging in size from that of a pinhead to that of a pea, each surrounded by a bright areola. These vesicles soon become pustules which dry into scabs that fall off and leave pits. The eye becomes photophobic and the lid the seat of an inflammatory œdema prior to the appearance of the eruption. The neuralgic pain also precedes the eruption, as a rule, and may outlast it a long time. The skin is hypersensitive during the attack, but later may be anæsthetic, or may present areas of anæsthesia. Ocular complications are of great importance and will be discussed under lesions of the cornea. The cutaneous eruption lasts only ten or twelve days, but the inflammation of the eye may continue for several weeks, and the eyeball may long be subject to attacks of neuralgic pain. The only disease with which we are liable to confound herpes zoster is erysipelas, but the limitation of the inflammation to an area supplied by a single nerve, the line of demarcation along the middle line of the face, the more intense neuralgic pain, the absence of an irregular, advancing, elevated margin, and the comparison of the groups of small vesicles with the large bullæ of erysipelas, make the mistake seem almost inexcusable. The pustules of impetigo are about as large as peas, but they are seldom seen on the lids, and they are not accompanied by the general symptoms

characteristic of herpes zoster. The bullæ of dermatitis herpetiformis are small and grouped, but the other signs of herpes are absent; those of pemphigus are large and scattered.

Discolorations

Many women have a slight dark discoloration of the lids, especially about the inner canthus, during menstruation. Brown spots sometimes appear on the lids during pregnancy, in the cachexiæ of cancer and tuberculosis, and in exophthalmic goiter. The bronzing of the skin in Addison's disease may be particularly marked along the margins of the lids. Little dark blue spots caused by grains of powder are common, the brown ones produced by bits of steel less so, and we seldom see a bluish stain as the result of the administration of silver. Small bright red spots that do not vanish on pressure may be symptoms of purpura hemorrhagica, but usually are congenital arterial angiomata, the larger of which, as well as the venous angiomata, are spoken of commonly as **birthmarks.** Venous angiomata are of a darker, bluish red, and have more diffuse outlines. Pigmented nævi are either smooth, soft spots of pigment, or hairy, wart-like elevations; usually they are stationary, or grow very slowly, but they may give rise to sarcomata. The skin may be clear and devoid of pigment in albinism, or clear whitish spots may be seen in it that have resulted from vitiligo, and sometimes it presents a peculiar piebald appearance that has been imparted to it by the abnormal pigmentation and cicatrization produced by the rare xeroderma pigmentosum. Much more often we see little flat elevations in the skin of the lids, commonly about the inner canthi in middle aged and old women, sometimes arranged symmetrically on each side; these are the benign tumors known as **xanthelasma,** or xanthoma, which grow very slowly and are not likely to cause any trouble aside from the disfigurement they occasion.

ATROPHY OF THE SKIN OF THE LID

This is seen most frequently in the aged, in whom the skin looks wrinkled, withered, and often yellowish, but it occurs sometimes in cachectic conditions and in cretinism. Occasionally we meet with a young person in whom the skin of the lid is so thin that it looks like a reddened bag of crinkled silk paper. This has been called by various writers **blepharochalasis, dermatolysis,** and **angiomegaly of**

the lids. The skin is very thin and allows the blood vessels to be seen through it. The actual extent of skin is increased and a ptosis may be caused, but this more commonly is simulated, as a careful inspection is likely to reveal that the margin of the lid is at its normal level. This trouble begins with an œdema of the upper lid that soon passes away and recurs. Its origin is unknown. A similar picture is produced sometimes by a lipoma, or by a hernia of orbital fat between the orbicularis and the tarsus, but such lesions present objects that can be felt by palpation. It has been stated that a hernia of fat may appear as a complication. Atrophy of the skin of the lid is the final result of scleroderma, is present in progressive facial hemiatrophy, and is one of the signs of general cutaneous atrophy, in which it may be congenital.

HYPERTROPHY OF THE SKIN OF THE LID

Hypertrophy is the result of some such inflammation of the skin as acne rosacea, eczema, scleroderma, elephantiasis, or rhinophyma, and is apt to be produced by repeated attacks of erysipelas. It forms a part of congenital hypertrophy of one side of the face, in which the skin is thick and soft, the upper lid in a state of ptosis, and then it often is associated with a plexiform neurofibroma and buphthalmos. Aside from the disease by which it is caused a general hypertrophy of the skin of the lid is of importance mainly because of the disfigurement and the increased weight of the lid that are produced, which are extreme in elephantiasis. The hypertrophy caused by scleroderma may be circumscribed or diffuse, but it always ends in atrophy.

DISORDERS OF SECRETION OF THE SKIN OF THE LID

Hyperidrosis points to a lesion of the sympathetic nervous system. Chromidrosis usually is met with in nervous and hysterical women and may occur in the outer half of the lid alone. All other disorders of secretion are symptomatic of general conditions in which the eye is not likely to be involved.

SORES ON THE LIDS

An open sore on the surface or margin of the lid, omitting for the present the small ulcers found in blepharitis, may be a broken down

pustule of small pox, chicken pox, herpes, or vaccine, a lupus or some other form of tuberculous ulcer, anthrax, a chancre, a chancroid, a serpiginous syphilide, a broken down gumma, an epithelioma, a diphtheritic ulcer, or the result of traumatism. The first three can always be recognized through the presence of other symptoms of the disease. A **vaccine pustule** is almost always on the margin, the ulcer has a clear red base, it is associated with great œdema of the lids and signs of a violent inflammation, but with slight constitutional symptoms, and a history of recent vaccination usually can be elicited. A vaccinated child may scratch his arm and then his eyelid, another person may touch the sore, or its dressings, and transfer the virus to his own eye, and even physicians have thoughtlessly rubbed their own eyes after performing vaccination and thus inoculated themselves.

An irregular ulcer which is without a hard elevated margin and with a base that is bluish, or covered with yellowish granulations, probably is **lupus,** or at least some form of tuberculous ulcer, but it may be a **serpiginous syphilide,** a **blastomycetic dermatitis,** or a **sporotrichosis.** The presence of similar lesions elsewhere usually aids in the diagnosis, but this can be made positive only by the bacteriological findings and the results of biological tests. The discovery of tubercle bacilli in the tissue or granulations marks the tuberculin test. The presence of other symptoms of syphilis, a positive Wassermann or Noguchi, and above all the finding of spirochætæ with no tubercle bacilli, is conclusive evidence that it is a syphilide. The detection of blastomycetæ or other fungi, particularly in the absence of syphilitic or tuberculous symptoms, renders it extremely probable that these are the cause of the ulcer.

Anthrax may start from a primary focus in the lid. The diagnosis is established by the extreme severity of the local inflammation, the very grave constitutional symptoms, the occupation of the patient, when it involves the handling of animal products, and by the finding of anthrax bacilli.

When the ulcer has sharply cut, elevated, and indurated margins, and a sloughy base we think of **chancre,** which often is hard to differentiate. The history is apt to be of little value, except in the way of exclusion of other lesions, as the infection may have come from a kiss, or the use of a soiled handkerchief or towel from two weeks to a month prior to the appearance of a little hard nodule on the margin of the lid, on the top of which an ulcer formed a

few days later. The only distinguishing characteristics of the sore at this time are its form, a peculiar hardness of its base, and the indolent swelling of a lymphatic gland, the preauricular if the lesion is in the outer part of the lid, the submaxillary if it is in the inner. A second chancre sometimes develops from contact at the corresponding place in the margin of the other lid. Toward the end of the second week a progressive, indolent swelling of the cervical glands gives evidence of the commencement of a general infection, and the secondary symptoms of syphilis appear in about the sixth week. The only way in which a positive diagnosis of chancre can be made in its early stages is by the finding of spirochætæ pallidæ in scrapings taken from its surface, but these are hard to detect and failure to find them may leave us in doubt until the appearance of the secondary symptoms. ***Wassermann's and Noguchi's tests are of no value*** at this time because the infection has not become generalized, or rather a positive reaction would go to show that the lesion probably was not a chancre.

A **chancroid** is met with rarely. As a rule it forms a yellowish, irregular ulcer with red, sharply excavated, but soft margins, which is situated usually at the edge of the lid, or at the canthus, where it is associated with a considerable degree of œdema and inflammation, and a painful, tender, and swollen preauricular gland. I have seen one case in which the sore on the lid did not extend to the margin and was productive of only a slight degree of local inflammation and swelling of the preauricular gland. The diagnosis was based on the appearance of the ulcer, the absence of any known traumatism, the exclusion of other forms, and the presence of chancroids on the penis of the patient, who was supposed to have infected his lid with his fingernail.

A deep ulcer that has a punched out appearance, a base which, though infiltrated, lacks the hardness of a chancre, and is accompanied by severe inflammatory symptoms, may be broken down **gumma.** The diagnosis generally is to be made from the history, the presence of other tertiary luetic symptoms, a positive Wassermann or Noguchi, and the exclusion of other forms of ulcer.

A **diphtheritic** ulcer has a gray coating over a dirty looking base, is ordinarily associated with a diphtheritic conjunctivitis, and can be diagnosed with certainty by the finding in it of Loeffler's bacilli.

A **superficial infected wound** may be of any size and shape, and may be situated anywhere on the lid, where it forms a traumatic

ulcer. The history, the absence of characteristics peculiar to other varieties, the presence of ordinary pyogenic microorganisms alone in scrapings taken from its surface, and its tendency to heal, ordinarily are sufficient for a diagnosis.

When we find one or more hard nodules on the margin of the lower lid near the inner canthus, possibly elsewhere, with the surface broken down so as to form a moist erosion, in an elderly person, we suspect an **epithelioma,** of which there are two clinical forms, the superficial and the deep. In the ***superficial*** form, which is the slower in its course and often is called rodent ulcer, a scab forms over the erosion so as to conceal a cancerous sore that develops with thick, elevated, hard, and nodular margins, and a base which exhibits papillary excrescences in some places and cicatrization in others. The healthy skin in the neighborhood may be thrown into folds by the contraction of this cicatrization. In the ***deep*** variety, sometimes called true epithelioma, an ulcer forms in the same way, but extends more deeply into the subjacent tissues of the lid, involves the conjunctiva, and threatens the integrity of the eye itself. The neighboring lymphatic glands rarely are involved in either form, and there may be said to be no metastasis. An endothelioma presents the same clinical picture and can be differentiated from an epithelioma only under the microscope.

Gangrene of the lid occurs in anthrax, sometimes in erysipelas, and rarely appears spontaneously in debilitated persons, when it is called noma. *Roemer* states that he has seen gangrene follow chicken pox. It has been caused by the application of ice directly to the lid, perhaps with the interposition of a piece of cloth, for which reason cold should never be applied to the eye in this way.

TUMORS OF THE LID

An **epithelioma** is a true tumor, although its clinical appearance is that of an ulcer, under which it has been described. It may start from a wart, but usually the history is that a little lump formed and then broke down, and in rare cases it has been known to develop in one of the glands of the lid, so in its early stages it is apt to form simply a lump. Hence for diagnostic purposes the term tumor of the lid must be used in its broadest sense, to include all localized swellings of whatever nature in its tissues, as these have to be differentiated from one another.

A lump at the margin of the lid, as large as a pea or smaller, that presents no signs of inflammation probably is a chalazion, but may be the nodule of a commencing epithelioma, a sarcoma, a chancre, a circumscribed gumma of the tarsus, a tuberculous node, a cyst, or an adenoma of the Meibomian glands. A **chalazion** is roundish, nodular, sometimes though not always adherent to the skin, feels tense on palpation, but is not tender, unless it is inflamed, and is intimately attached to the tarsus, with which it is movable. An inflamed, suppurating chalazion hardly needs to be differentiated from a hordeolum, from which it differs for practical purposes only in the history of a lump of long standing. The nodule of an **epithelioma** is met with almost exclusively in old people, usually on the lower lid near the inner canthus, and is not attached to the tarsus, as a rule. We do not often see it in the stage in which it is liable to be mistaken for a chalazion, because old people are apt to think little of such a lump until its surface has become eroded. A **sarcoma** may be hard to differentiate clinically unless it is pigmented, or is separate from the tarsus. It is quite rare; according to *Veasey* only about fifty cases are on record. A melanosarcoma is likely to be recognized readily as it forms a gray, or black brown tumor, and appears gray black through the conjunctiva if it is situated deeply. This form is quite apt to start from a pigmented nævus. A leucosarcoma may start in the tarsus, or between this and the skin, and then it may be differentiated in some cases by a growth that is more rapid than that of a chalazion, and by a necrotic destruction of its interior which may be accompanied by hemorrhages, but a histological examination often is necessary to a positive diagnosis. It occurs at all ages, the round cell and the spindle cell varieties seem to be about equally common, the mixed cell form less so, while the plexiform angiosarcoma and the cylindroma are rare. A **chancre** is hard, is situated in the skin, not in the tarsus, and is movable over the latter. A circumscribed **gumma** of the tarsus may have about the same situation as a chalazion, may be of the same shape, size, and consistency, may present the same appearance on both the inner and the outer sides of the lid, and have the same history of a slow, painless development. Fortunately it is rare. Such a tarsitis develops in both hereditary and acquired syphilis, and a chalazion may form readily in the lid of a person suffering from syphilis. The only way in which the differentiation can be made is through the effects produced on the growth by internal medication. Whenever

anything suggests that the growth may be syphilitic it is wise to give the patient large doses of potassic iodide for a few days; it will grow smaller if it is a gumma, but it will not be affected if it is a chalazion. A diffuse ***gummatous tarsitis*** usually is accompanied by inflammation, but if this is absent it resembles a giant chalazion, a broad flat swelling produced by a granulating inflammation of a number of adjoining Meibomian glands; again the diagnosis can be made only by watching the effect produced by large doses of potassic iodide. A ***tuberculous tarsitis*** usually is secondary to tuberculous lesions of the skin or conjunctiva. An endogenous primary infection of a Meibomian gland is very rare, and when it occurs it is apt to cause a swelling that cannot be told from a chalazion by its appearance. A local reaction to an injection of tuberculin may make the differentiation, but sometimes the diagnosis is impossible except by a histological examination of the tissue. A cyst of a Meibomian gland appears through the conjunctiva to be more translucent than a chalazion. **Adenomata** of the Meibomian glands are said to cause the lid to become stiff and boardlike, and to present nodular masses which have a yellowish color on the conjunctival surface. Similar tumors have been found in Moll's, Krause's, and Zeiss's glands, where they may be difficult to differentiate at first from commencing carcinomata, but are distinguished finally by their course, as they do not break down.

When a patient has been seized suddenly with a painful feeling of tension in a diffusely red and œdematous lid, we may be able to feel by careful palpation a small, hard, exquisitely tender nodule near the margin. If it is free from the tarsus it is an **external hordeolum,** a staphylococcic inflammation of a sebaceous gland, or if it is quite superficial we may call it a **boil** or furuncle, which is a similar inflammation of a sebaceous or sweat follicle. If it is seated in the tarsus a Meibomian gland is inflamed and we have a **hordeolum internum.** All three are called **styes.** The nodule grows larger, the pain becomes severe, the overlying skin becomes very red and finally exhibits a yellow point which indicates that it has broken down into pus and is about to rupture. The least common variety is the hordeolum internum, and in this the course is longer than in the others, the symptoms are more severe, the conjunctiva becomes œdematous, and the clinical picture is that of a deep seated abscess of the lid.

An inflamed swelling which contains pus is an **abscess.** It may

arise from a suppurating hordeolum, or from the entrance of pus agents into the subcutaneous tissue through a lesion in the skin, or it may originate from a cellulitis of the orbit, a periostitis of the margins of the latter, an empyema of the frontal sinus, or a purulent ethmoiditis. When the cause is not evident it should be sought for diligently.

A **carbuncle** may be termed a conglomerate of furuncles attended by a severe constitutional disturbance, which includes a beating headache, and is characterized by a brawny hardness and a sloughing of the tissues involved. Usually it appears in the region of the brow, and after it has ruptured it may present many perforations. The condition is one which is quite grave as it is apt to cause not only gangrene of the affected tissues, but also a thrombosis of the veins in the orbit, and death through a purulent meningitis and pyæmia.

On rare occasions we may feel a circumscribed, soft, elastic, lobulated tumor that extends more or less back into the orbit and is usually if not always congenital; this is a **lipoma.** Symmetrical lipomata, situated between the orbicularis and the tarsus, sometimes produce a picture that resembles blepharochalasis, but the tumors can be felt.

Much more commonly we find a more or less spherical congenital growth over which the skin is freely movable in the upper lid, either at the inner canthus, in front of the fronto-maxillary suture, or at a point in the upper outer margin of the orbit that corresponds to the fronto-malar suture, less often elsewhere, but always at a place where sutures or fissures existed during fetal life. Ordinarily such a tumor is a **dermoid,** but it may be a hernia cerebri, a term that includes both meningocele and encephalocele, or a cyst that has been formed by an obliteration of the pedicle of a meningocele. The differentiation of these conditions, which have several symptoms in common, is of importance. All may be attached firmly to the bone, or connected with it by a pedicle, and in all we may be able to make out by palpation an opening in the bone at the site of the suture. When the tumor is felt to pulsate, and can be made smaller by pressure, which causes at the same time such signs of increased brain pressure as nausea, vomiting, vertigo, and convulsions, it is a **hernia cerebri** and the brain symptoms have been caused by the return of a part of its contents to the cranial cavity. A cavernous angioma can be made smaller by pressure, but the diminution in size is not

attended by brain symptoms, and its return to its former size when the pressure is removed is immediate, while a hernia cerebri regains its size much more slowly. When no pulsation can be felt, and the size cannot be reduced by pressure, the tumor may be a dermoid, or a cyst formed by the constriction and obliteration of the pedicle of a meningocele. Both may feel firm on palpation, but in a cyst we may be able to detect fluctuation; otherwise we are apt to remain in doubt.

Little translucent tumors along the margin of the lid, perhaps as large as peas, are **retention cysts** due to occlusion of the excretory ducts of the glands of Moll. Retention cysts of the sebaceous glands form little, round, yellowish swellings, and are called **milia;** they are more common on the lower than on the upper lid and sometimes resemble xanthoma when they are grouped. Congenital milia probably are caused by a snaring off of bits of the epidermis into the corium during fetal life. Cutaneous horns are recognized readily, and warts present the same characteristics as when on other parts of the body. The latter may give rise to carcinoma in old people.

A little tumor which is attached to the skin of the lid by a pedicle and hangs down like a pouch, is a **fibroma.** Another name is simple molluscum, but this is not good, for it is related in no way to the much rarer **molluscum contagiosum,** a smooth, semiglobular, yellowish or pinkish tumor, that varies in size from that of a hempseed to that of a pea, and has a central depression in its top from which its soft, whitish contents can be made to exude by pressure. This tumor generally is met with in numbers along the margins of the lids of uncleanly children, where an older one frequently may be seen to be surrounded by a group of more recent, smaller ones, Whenever one appears on the margin of a lid another soon develops on the other, and *Retzius* proved it to be contagious by inoculation.

A red or bluish, roundish tumor of the lid that is easily compressible, but regains its size as soon as pressure is removed, and swells when the blood is driven into the face, whether by position or by the emotions, is an **angioma.** It is a mass of enlarged blood vessels which grows slowly and may reach an enormous size. Usually it is congenital and may form simply a red spot, which commonly is called a birthmark, a little blue growth, or a large lobed tumor. When it contains large spaces it is called a ***cavernous angioma.*** A congenital, elastic, diffuse swelling that resembles a

venous angioma but is pale and bears more resemblance to a cyst, is a ***lymphangioma,*** which consists of hollow spaces lined with epithelium. Very large lobulated tumors of similar nature are known as congenital **elephantiasis lymphangiectatica.**

Quite rarely we meet with a rather soft congenital tumor in the upper lid from which hard cords may be traced by palpation; it is not painful, as a rule, but may be tender in places. This is a **neurofibroma,** and generally is associated with a hypertrophy of the skin that causes ptosis, frequently with buphthalmos.

Sometimes the lids are pressed out by tumors beneath them, or perhaps invading their tissues to some extent, as in affections of the lacrimal gland, and in leucocythæmia. The former are described under diseases of the lacrimal organs, and the lymphomata of the latter disease will be discussed under tumors of the orbit.

BLEPHAROPHIMOSIS

When the palpebral fissure is shortened by an adhesion along a part of the margins of the upper and lower lids the condition is known as blepharophimosis. The adhesion itself is called an **ankyloblepharon.** This condition is congenital in rare cases, in which it may have been caused by an epithelial adhesion, or by the union of the lids through connective tissue for a greater or less distance from the outer canthus, but more often it is due to a cicatricial contraction of the conjunctiva, or to the adhesion of the margins of lids which have been made raw by ulcers or burns. A severe burn may cause the lids to agglutinate throughout their entire extent and produce acquired **cryptophthalmos,** which usually is associated with an extensive symblepharon. Congenital cryptophthalmos, produced by a total ankyloblepharon, generally epithelial, is common in many animals, but is rare in man.

The growth of the lids is restricted together with that of the neighboring tissues after enucleation or atrophy of the eye in childhood, and this is likely to result in a blepharophimosis in the adult. Sometimes we need to determine whether a blepharophimosis is real or not, for atony of the external palpebral ligament may counterfeit a stenosis of the palpebral fissure, and the latter may seem to be shortened by a vertical fold of skin that covers the outer canthus. Usually such a fold can be drawn back and the fissure revealed to be of its normal size, but its skin may become irritated by constant

wetting in a conjunctivitis so as to be hard and contracted horizontally, while a blepharospasm may be excited, and we be led to think the fissure too short until the seeming blepharophimosis passes away with the recovery from the conjunctivitis.

LAGOPHTHALMOS

When the lids lie wide apart and can be closed with difficulty, if at all, we say that the patient has lagophthalmos. In very rare cases this condition may result from a congenitally too short upper lid, or from a spasm of the levator palpebræ, but much more commonly it is due to a paresis of the orbicularis, to ectropion, to exophthalmos, to an enlargement of the eyeball, to a tumor that separates the lids, or to a very grave general disease in which the sensorium is so benumbed, or the sensitiveness of the cornea so impaired that the patient no longer winks. The differentiation of these conditions ordinarily can be made at a glance. Sometimes the eyeball protrudes so much in high myopia that the presence of lagophthalmos is suggested, but this can always be excluded if there is no circumscribed patch of redness of the bulbar conjunctiva on each side of the cornea, which will always form at the place habitually left uncovered by the lids. The consequences of this condition will be discussed under keratitis e lagophthalmo.

ECTROPION

Eversion of the lids is ectropion. It may be very slight, so great that the entire inner surface of the lid lies exposed to view, or of any intermediate degree. The diagnosis is made readily except in very slight cases, when attention may need to be drawn to it by an epiphora and a slight thickening of the conjunctiva near the margin of the lid. Differentiation deals mainly with the causes, which are diverse.

A cicatrix that makes traction on the anterior part of the lid is a sufficient explanation; so is a cicatricial fistula that leads down to carious bone in the vicinity, if similar traction is exerted. This is **cicatricial ectropion,** the degree and extent of which varies according to the size and position of the scar. The highest possible degree may be produced by an extensive burn of the face. I have seen the upper lid everted until its lower margin coincided with the line of the

eyebrow, and the margin of the lower drawn down nearly to the level of the ala of the nose, in a child that had fallen into a fire and burned the entire side of the face. The cicatricial contraction that follows lupus can produce almost if not quite an equal degree. Smaller cicatrices may evert only a portion of the lid, when the ectropion is said to be partial. That which is produced by fistulæ is partial and usually is about the outer canthus.

When there is no explanatory cicatrix we look for other causes. The weight of a tumor may cause a lower lid to turn out. A relaxation of the margin which allows the lower lid to droop is seen occasionally in facial palsy; this is due to paralysis of the orbicularis, is known as **paralytic ectropion,** and is differentiated through a paresis of other muscles on the same side of the face. The strength of the orbicularis fails along with that of other muscles in old people, while at the same time the skin of the lids becomes relaxed. This relaxation may pass the point at which the lower lid is supported against the eyeball, or the weight of the lid may be increased by a chronic conjunctivitis sufficiently to allow the margin to fall away a little and produce a **senile ectropion.** All forms tend to grow worse and many points in the course of the senile variety are common to all. As soon as the palpebral conjunctiva is allowed to remain in contact with the air it becomes hyperæmic, swollen and inflamed, presses the lid out still farther, increases its weight, and so renders the ectropion worse. The lacrimal punctum is turned outward, the tears overflow and cause a dermatitis which makes the skin of the lid heavier, and this helps to drag it down. The conjunctiva hypertrophies, dark red elevations appear in it, and, if the condition persists long enough, its surface becomes dry, lusterless, covered with scabs, and the epithelium becomes almost horny. The ectropion sometimes met with in a chronic blepharoconjunctivitis admits of a similar explanation; the tissues of the lid become relaxed from some cause, perhaps ill health, and then the conditions present are favorable for its development.

When we try to open the eye of a child it sometimes happens that his lids evert; this is **spastic ectropion,** and is produced in a different way. The tense, elastic skin of the lid tends to draw the margin of the latter toward that of the orbit, and when a considerable swelling of the conjunctiva presses the margin outward from the eyeball a strong effort on the part of the orbicularis to keep the eye closed will evert the lid. The upper lid is the one generally affected, but

sometimes both, or the lower one alone, turn inside out in this manner. Once this has happened the peripheral fibers of the orbicularis contract spasmodically and keep the lid in this malposition, a venous engorgement ensues and the conjunctiva forms a thick red swelling; then it may be difficult to turn the lid back, or to keep it in its proper place, for sometimes the swelling is so great that the spasm of the orbicularis produces the eversion spontaneously. This condition is seen most often in phlyctenular keratitis, but is met with occasionally in trachoma. An ectropion is produced in a similar manner when the tarsus is given an abnormal position by a tumor of itself or of the conjunctiva, by a protrusion from the anterior surface of the eyeball, or by exophthalmos. The causal lesion is evident in these cases. A notable example of this form can be seen sometimes in a patient who is suffering from exophthalmic goiter, when he is seized with a very painful spasm of the orbicularis which everts the lids, and perhaps dislocates the eyeball. Congenital ectropion is a rare malformation.

ENTROPION

When a patient complains that his lashes irritate his eyes he may have a few wild hairs, or some fine, white maldirected ones at the inner canthus, but the chances are that he has entropion. If the lashes are arranged normally along the outer edge of the margin of the lid, but are directed nearly parallel to the surface of the eyeball, they will touch the latter occasionally or in places, and we are dealing with a very mild degree, or an early stage of entropion, even though the inner edge of the margin of the lid may still be seen. If the lashes are distorted and disarranged the patient has trichiasis also, a combination which we meet with very commonly. When the lashes rub against the eyeball and the inner edge of the margin can no longer be seen the condition is further advanced, and when the lashes are hidden, but can be brought into view by traction on the skin of the lid so as to turn the margin out, it is extreme. Congenital entropion is very rare.

The two varieties of entropion ordinarily met with are the spastic and the cicatricial, the differentiation of which is very important, as they are not amenable to the same treatment.

Spastic Entropion

Spastic entropion is met with commonly in old age and therefore has been called **senile,** but it occurs at all ages. It is produced by the contraction of the orbicularis, but as the strongest contraction of this muscle cannot produce it in a normal lid, the main factor of its production must be sought for elsewhere. We find it in a relaxed condition of the skin, which ordinarily exerts sufficient tension to keep the margins of the lids almost perpendicular to the surface of the eye. When for any reason, be it a cicatricial contraction on the part of the conjunctiva, or a loss of tone on the part of the skin, the outer edge of the margin overrides the inner in the act of winking, so that the inner edge makes an acute angle with the surface of the eye, the traction of the peripheral fibers of the palpebral portion of the muscle tends to make the margin turn in. Lack of normal support on the part of the eyeball produces the same effect, as may be seen after an enucleation, and the eyes of old people are apt to be sunken. Hence the predisposing factors to spastic entropion are a relaxation of the tension of the skin of the lid and a recession of the eyeball; the actuating factor is a contraction of the orbicularis. This form frequently is excited by the bandaging of an eye for a few days, as after the extraction of a cataract.

Cicatricial Entropion

The first thing we do in most cases in which we find the margin of the lid to turn in is to evert the lid and examine its conjunctival surface, for if the conjunctiva has been replaced by cicatricial tissue the contraction of this is ample to account for the condition. Cicatricial entropion occurs most often after trachoma, but sometimes it follows burns, or pemphigus. As the cicatricial conjunctiva contracts the inner edge of the margin is drawn back and rounded off until the intermarginal space is obliterated. This brings the lashes to rub on the surface of the eye, even when they are intact, which rarely is the case. In the great majority of the patients the tarsus has been deformed and the bed of the cilia diseased by the primary affection, so that the entropion is complicated by trichiasis. The symptoms produced are the same as those of the spastic variety, but are much more intense:—pain, a feeling as though a foreign body were in the eye, lacrimation, photophobia, conjunctivitis, keratitis with the formation of ulcers, or of inflammatory opacities.

These two varieties often can be differentiated by making vertical traction on the skin of the lid; if this restores the lid to its normal position temporarily the entropion is spastic, otherwise it is cicatricial.

THE EYELASHES

The eyelashes are placed close together in a double or triple row along the outer edge of the margin of the lid, curve upward on the upper, downward on the lower, so that they do not interlace when the eye is closed, and each lash tapers to a point. Those on the upper lid are longer and twice as numerous as those on the lower. Each lash lives four or five months and then is cast off to be replaced by another. Sometimes rows of supplementary cilia are found growing from the posterior part of the intermarginal space in lids that otherwise are normal, and usually are described as very fine hairs that occupy the site of the Meibomian glands. I have had the opportunity to observe for a number of years a case in which a row of well formed lashes, of the same color as the others and curving with them, occupies this site in all four lids and has caused no trouble. This is **distichiasis.** It is congenital and unimportant, except for the fact that it may be mistaken for the condition of trichiasis in which the distorted lashes happen to form a second row. *Westhoff* is quoted by *Ball* as saying that sometimes it appears to be hereditary. Occasionally an irregular and confused growth of lashes appears in old people from rudimentary or pathologically altered hair germs. A very rare condition is a hereditary arrest of the growth of each lash at certain points so that each looks beaded and is brittle between the beads. If the lashes are normal in every way except that they are clubbed instead of ending in points, it is probable that they have been cut or singed.

The lashes retain their **color** in age longer than the hairs of the scalp, but finally they become gray. White lashes may be congenital, and sometimes this is true of only a part of them, but colored lashes have been known to turn white rather suddenly in sympathetic ophthalmia, in trigeminal neuralgia, after operations on the eye, and even when no cause could be assigned. *Roemer* says that this has occurred as the result of a fright, and imagines that the effect of the nervous and circulatory disturbance is to deprive the hair papilla of its power to form coloring matter, and to cut off the supply of pigment from the hair trunk.

Some or all of the lashes may be absent. Congenital **alopecia** forms a part of a universal alopecia due to a fault of development. More often absence of the lashes is due to a cicatrix, or to one of the diseases included under blepharitis, and when it is permanent it shows that the papillæ have been destroyed. Sometimes the lashes fall out after an attack of an acute infectious disease, like typhoid or scarlet fever, in syphilis, in chronic arsenical poisoning, in exophthalmic goiter, and after removal of the thyroid.

TRICHIASIS

The subjective symptoms of trichiasis are the same as those of entropion, for they are caused in both by the scratching of the surface of the eye by the lashes. It is quite exceptional to meet with a case of pure trichiasis in which we find the margin of the lid in its proper position, but with a lot of deformed lashes scattered irregularly over the intermarginal space, their points extending in every direction, many of them toward the eyeball. With a magnifying glass and oblique illumination we are able to make out more that are small and colorless, but quite as capable of irritating the eye as the larger ones. The appearance of all these lashes under the magnifying glass is such that it has been compared to that of a tangled forest. The trichiasis may involve the whole, or only a part of the margin, may affect any one or all four of the lids, and questioning will almost certainly elicit a history of a long standing blepharitis. In the great majority of cases the trichiasis is associated with entropion, the lashes are pressed more firmly on the surface of the eyeball, and the irritation is more intense. The cornea may become ulcerated where its epithelium has been scraped off, or the epithelium may become indurated, thick, opaque, and finally be covered by dense vascular opacities.

BLEPHARITIS MARGINALIS

When the margins of the lids are red and swollen we say that the patient is suffering from blepharitis marginalis, a term which is inclusive of a number of local diseases. We may divide it clinically as well as pathologically into three varieties:—hyperæmia, seborrhea, and ulcerative blepharitis.

Hyperæmia of the Margin of the Lid

It is well known that the eyes are apt to "swim in tears," the lids to be hot and heavy so as to impart a tired, sleepy aspect to the face, to itch and burn, causing the patient to rub them and distracting his attention from his work, and their edges to be red, after a carouse with overindulgence in alcoholic liquor, but it is not so well known that the *same symptoms may be produced in certain persons by such slight external irritations* as smoke, wind, or a bright light, by eyestrain, by the emotions, and by severe bodily exertions. Yet this is true, and the unfortunate victims are apt to be misjudged harshly as habitually dissipated. Such a condition is not only an impediment to the enjoyment of innocent pleasures by persons who suffer in this way, but has proved a serious obstacle to success in business. A careful differentiation of the cause of a hyperæmia of the margins of the lids is imperative. In a doubtful case we may get some help from a chemical analysis of the contents of the stomach, where this shows the presence or absence of alcoholic gastritis.

During an attack of acute hyperæmia we find the margins of the lids to be very red, with many minute, bright red vessels, and to be slightly swollen, but to present no sores or scabs, no localized foci of inflammation, and no scales, as a rule. The palpebral conjunctiva is injected, the lacrimal secretion is increased. Repeated attacks are apt to result in a chronic condition in which the margins of the lids are constantly red, thick and heavy, with large, distended vessels ranging in color from red to violet. Such patients suffer a great deal from photophobia and exercise much care in protecting their eyes from the light. We have to ascertain the cause as best we can from the history and habits of the patient, as well as from an examination of the eyes for eyestrain.

Seborrhea

Not infrequently when a patient complains of itching and burning of the margins of the lids we may be able to see little or nothing abnormal at first, except perhaps some little whitish scales about the roots of the lashes. If we rub the lashes these scales become loosened, part fall off, and the rest cling like powder to the lashes and the margin of the lid. After removing these with a bit of wet

cotton the skin is seen to be reddened, but not ulcerated. Once in a while crusts that glue the lashes together will be seen, but they do not cover ulcers and they must not be confounded with the crusts of dried mucus and pus that glue the lashes together in acute conjunctivitis. The scales and crusts of this **seborrhea sicca** consist of dry sebum with cast off epidermis, and cover colonies of fungi, which probably cause the disease, located on the surface and in the excretory ducts of the glands. The lashes are apt to be loose and some fall out when we rub them, but usually they grow in again unchanged; it is only when the condition has lasted a long time that they become distorted or permanently lost as the result of the extension of the disease to the hair papillæ. Sometimes the excessive secretion of sebum from the sebaceous glands is so great that it does not dry into scales, but gives the lashes an oily appearance, although the other symptoms remain about the same; then the disease is called **seborrhea oleosa.**

Although this disease probably is parasitic in origin, its development and persistence are favored by refractive errors, especially hypermetropia and astigmatism, which increase the congestion of the margin of the lid, a fact that needs to be borne in mind in the treatment of nearly all marginal affections. The irritation may be caused also by **crab lice.** *Lawson* says that pediculi capitis have never been known to locate in either the brows or the cilia, even when they were swarming on the scalp. The scales formed at the roots of the lashes are dark from the nits contained in them, and black nits are to be seen on the lashes themselves, so the condition is recognized easily with the aid of a magnifying glass. We never ascribe such a condition as this to the **demodex folliculorum,** which often is to be found on the roots of the lashes, or in the sebaceous follicles, because this parasite very rarely if ever induces any symptoms in man.

Blepharitis Ulcerosa

When we find ulcers on the margin of the lid beneath the crusts we have to deal with a follicular or perifollicular inflammation of the hair follicles which is commonly referred to as blepharitis ulcerosa. The largest number of cases are eczematous in their nature and are met with in ill conditioned children; a smaller number appears in adults, among whom sycosis is more common, though eczema sometimes occurs.

The first stage of an **eczematous inflammation of the margin of the lid** presents a hyperæmia with many little red nodules about as large as pinheads, which quickly become vesicles, forming the second stage. The third stage follows rapidly in which the vesicles change to pustules, which soon rupture, leave erosions, and usher in the fourth stage. The first three stages are apt to escape observation because of the rapidity with which they develop, but the fourth lasts a long time and is the one commonly seen. The margins of the lids are swollen, thick, and covered with crusts that glue the lashes together. Near the crusts we often see pustules, or deep crater-like ulcers with an eyelash protruding from each, as in sycosis, but the surrounding inflammation negatives the probability of the latter; in any case of doubt we should pull out one of these lashes and examine its root under the microscope in search of a tricophyton. As soon as the crusts are removed a depressed raw surface that bleeds easily is revealed.

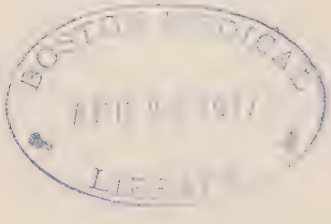
The course of an eczematous blepharitis is very slow, but its ill results may be extreme. The lashes fall out because of the injury done to their follicles, and when the disease is neglected they may not return at all, leaving alopecia, or they may return in faulty positions and present the disarranged and distorted appearance described under trichiasis. At the same time the margin of the lid sometimes becomes inverted or everted, producing entropion or ectropion. Perhaps the most familiar result in a neglected case is a smooth, thick, glazed, everted margin, bordered on the inner side by red, and more or less destitute of lashes, which is a hideous deformity that produces epiphora through its interference with the normal conduction of the tears, with a consequent irritation of the skin. The result is even more horrible when the inner edge of the margin is eroded so as to produce entropion. When the inflammation subsides the formation of crusts lessens and epithelium forms over the erosions, but this is desquamated for some time in the form of whitish scales. This is the stage of healing.

The differentiation of the various diseases that are classed together as blepharitis ulcerosa is not so very difficult. Crusts are apt to mat the lashes together in acute conjunctivitis, and sometimes do so in seborrhea, but ulceration is never found beneath them. In **eczema** we are apt to find the disease elsewhere on the skin, the conjunctiva is likely to be affected, and conditions that aggravate the trouble often can be found in the nasal cavity. **Sycosis** usually is discrete,

though in bad cases the entire margin may be involved and present a picture that is not typical. A man with pustules about the bases of the lashes, or with ulcers having a lash protruding from each, probably has ***sycosis parasitica,*** or barber's itch, especially if he has a similar eruption elsewhere on the face, and this diagnosis is made certain if we find the tricophyton tonsurans in the swollen root of an eyelash epilated from one of the pustules or ulcers. If the microscope reveals staphylococci, or some other microorganisms, but no tricophyton, the rest of the skin is healthy, and there are no signs of eczema, the disease is ***sycosis coccogenes.*** Very rarely we may see a yellow dry, dimpled scab on the margin of the lid, beneath which the skin looks peculiarly lustrous and moist; this is the characteristic appearance of favus.

LEPROSY

According to *Lyder Borthen* the nodular or tubercular form of leprosy is quite apt to start in the eyebrows, where the whitening and the falling out of the hairs may be the first, and sometimes for years the only sign of the disease. The skin of the lids may be infiltrated, or may present large nodules symmetrically arranged along the free margin. The lashes are affected in the same way as the eyebrows, they become white and fall out. The maculo anæsthetic form of the disease is said to appear occasionally in the same location but not as commonly as the nodular. Nodules are said to appear in the conjunctiva, which usually is inflamed, and to invade the cornea, where they develop beneath the epithelium and have been found to be composed mainly of accumulations of leprosy bacilli. An interstitial keratitis is said to be not uncommon. Sometimes multiple, rarely single nodules appear in the iris and partly fill the anterior chamber. The diagnosis is made by incising an infiltrated place in the skin, squeezing out some of the juice and examining it for leprosy bacilli.



CHAPTER III

The Lacrimal Organs, Lymphatic Glands, and Accessory Sinuses

THE LACRIMAL ORGANS

The most common symptom referable to the lacrimal organs is a superabundance of tears in the conjunctival sac, which we call **lacrimation** when it is due to a hypersecretion of fluid by the lacrimal gland, and **epiphora** when it is caused by an obstruction to the outflow of the tears somewhere in the lacrimal passages. Strong emotions, such as sorrow, joy, and anger, occasion temporary attacks of lacrimation, but when these emotions are absent, or the weeping is of a constant character, we are apt to seek for a pathological cause. The first step toward a diagnosis then is to ascertain whether lacrimation or epiphora is present. Lacrimation is apt to cause a patient to blow his nose frequently, or to cause fluid to drip from it, but such symptoms can be produced by a cold in the head when the patient has epiphora. A positive method of differentiation is to instill a drop or two of a solution of fluorescin into the conjunctival sac and have the patient blow his nose a few minutes later; if the discharge from the nose presents the characteristic stain of fluorescin the tears are following their regular course down the lacrimal passages and the lacrimal gland is secreting so much fluid that the capacity of these passages is overtaxed, but if the nasal discharge is not stained the outflow of tears is blocked, and the case is one of epiphora.

The usual cause of lacrimation, aside from the emotions, is irritation of the terminal filaments of the trigeminus, such as is produced in the eye by wind, or smoke, or by the presence of a foreign body in the cornea, and in the nose by inhalation of the fumes of ammonia. It is a symptom common to all superficial inflammations of the eye in which the sensitiveness has not been abolished. A dressing soaked with tears after an operation on the eye is indicative of some serious

irritation or commencing inflammation, provided that neither lacrimation nor epiphora existed previously. Occasionally a case of lacrimation cannot be explained by any irritation in the eye or the nose, and then it is well to remember that it may be one of the early symptoms of tabes dorsalis, or of exophthalmic goiter. Paroxysms of weeping that did not seem to be connected with the emotions have been described as occurring in tabes, anæmia, myxœdema, and pregnancy; in all such cases the lacrimation must be referred to a direct or a reflex action on the central nervous system. Curious forms mentioned in literature have been seen on arising in the morning, in the evening, or during mastication; when a local irritation can be excluded such cases as these must be referred to the same cause. Lacrimation occurs also in trigeminal neuralgia, but in this disease it is hard to exclude the psychical effect of pain.

The opposite condition, that of **subnormal secretion** of the lacrimal gland, is not so easy to recognize, for the eye remains moist after the function of the gland has been abrogated so long as the mucous glands of the conjunctiva continue to functionate, but it is present when no lacrimation can be produced by irritation of the cornea, or by the inhalation of the fumes of ammonia. In such a case the eye does not weep in response to emotion. The amount of the lacrimal secretion is subnormal when the gland has been rendered atrophic by the cicatricial occlusion of its excretory ducts, in dacryops, in Mikulicz's disease, in some cases of facial palsy, and in neuroparalytic keratitis.

The lacrimal secretion is spread over the surface of the eyeball and guided to the lacrimal lake by the movements of the lids; this guidance may be disturbed not only by a paresis of the orbicularis, but also by any malposition or mutilation of their margins. This lack of guidance constitutes an obstruction, so we have epiphora in facial palsy, ectropion, entropion, and when the margin of the lid is deformed in any way, even though the puncta may not be removed from the lacrimal lake. This withdrawal of a punctum usually is assigned as the cause of the epiphora that attends ectropion and entropion, and unquestionably does constitute a serious obstruction, but as two puncta plunge into the lake, one belonging to the upper, the other to the lower lid, this hardly seems to account for such a degree of epiphora as we often see in a slight ectropion of the lower lid alone, while the failure of the lower lid to do its share in the conduction of the tears is competent to explain their

accumulation along its margin and their overflow more satisfactorily. This is the explanation of the epiphora that exists when the margins of the lids have been mutilated in such a way that the puncta remain in their normal positions. In entropion the irritation produced by the lashes causes lacrimation in addition to the epiphora, and we are apt to speak of the former alone in describing the symptoms of such conditions.

One or more of the puncta may be ***absent*** congenitally; this usually, but not always, causes epiphora. More often we find the puncta ***stenosed,*** or occluded by a foreign body, such as a loose eyelash, one end of which has entered the opening. Stenosis may result from traumatism, burns, or severe inflammation, but more commonly from a blepharoconjunctivitis. Such conditions usually are apparent on careful inspection.

The canaliculi may be congenitally ***absent,*** or may become ***occluded.*** The presence of a scar may show that one has been cut across and now is closed by a cicatrix. The end of a lash protruding from the punctum tells its own story. A firm mass to be felt somewhere along its course informs us that it is closed by a foreign body; it matters little whether it is a dacryolith, a fungous growth, or some other substance. It is more difficult to diagnose a stricture at the point where the canaliculus opens into the lacrimal sac, but when epiphora cannot be accounted for by a fault of the lids, the punctum and most of the canaliculus are patent, there is no swelling or fullness over the region of the sac and pressure there does not produce the slightest regurgitation, we may be justified in deciding that a stricture exists at this point, especially if there is a history of antecedent probing. A **mucocele** of the canaliculus sometimes is to be seen as the result of a stricture at both ends.

DACRYOCYSTITIS

Much more frequently the stricture is situated farther down, at one end or the other of the nasal duct, and the epiphora then forms one of the symptoms of dacryocystitis. We find a fullness or swelling over the region of the sac, which can usually be reduced by pressure, the reduction being accompanied in most cases by a regurgitation of fluid from the punctum. If this fluid is watery or consists of mucus we call the condition catarrhal, if pus appears we say it is purulent. In some very mild cases of **catarrhal** dacryo-

cystitis there is no regurgitation although the fullness is reduced by the pressure, because the contents of the sac have been driven down into the nose; the normal passage of the tears through the nasal duct is blocked, but the obstruction is insufficient to resist a slightly increased force. The only symptoms present in such a case are the epiphora, a fullness over the lacrimal sac, which may be slight, and sometimes an obstinate chronic conjunctivitis.

Catarrhal and **chronic purulent dacryocystitis** are intimately related. The primal cause in most cases is a disease of the nasal mucous membrane that either blocks the lower opening of the nasal duct, or extends upward in its mucous membrane and forms a stricture at its junction with the sac. The tears accumulate in the sac, which becomes distended, the pyogenic germs found on the conjunctiva are carried down into it, where they remain and accumulate. These germs may continue to die off for a long time until some virulent ones enter, settle in the mucous membrane and multiply, when the type changes to that of a chronic purulent dacryocystitis. Finally the pus agents pass through the mucous membrane, enter the surrounding tissues, and inaugurate an acute purulent dacryocystitis, or abscess of the lacrimal sac.

Although this is the usual history, it is not the invariable one. Sometimes we meet with a mucocele of the sac that is due not to stricture, but to an ***atony*** of its walls, particularly in old persons. Occasionally we see an infant with a dacryocystitis due to a ***delayed opening*** of the lower end of the nasal duct; this disappears spontaneously in a few days, as a rule. Other conditions that may produce dacryocystitis are a congenital malformation of the nasal duct, fracture or caries of its bony walls, violent and injudicious probing, occlusion by a polyp or other tumor that springs from the mucous membrane of the lacrimal sac, a dacryolith or some other foreign body, or the pressure of a tumor from the nose, antrum, or base of the skull. It is possible for the lacrimal sac to be so occluded by the pressure of a tumor as to produce epiphora without dacryocystitis, but this happens very rarely. We must also look for evidence of any former operation on the lacrimal passages, as epiphora may follow, or not be cured by certain ones.

When a patient who has had lacrimal trouble, even though nothing more than a persistent epiphora, is seized with headache, fever, intense pain in the region of the lacrimal sac, which is red, swollen, and exquisitely tender, with a considerable degree of œdema of both

lids, which is most marked in the inner part of the lower one and extends down along the side of the nose, the diagnosis of **acute purulent dacryocystitis** is pretty certain, but in the absence of such a history we must question whether the trouble may not be an abscess in the subcutaneous tissue about, though not starting from the sac, which is possible, or the rupture of an empyema of the ethmoid, or of the frontal sinus into the tissues above the sac. A boil may occur in this location, but can be differentiated easily, as a rule. The first thing to do is to determine whether the lacrimal passages are open or closed. The best way to do this is to instill a drop or two of a solution of fluorescin into the conjunctival sac and to examine the contents of the nose for its stain a few minutes later. This is much gentler than an attempt to syringe fluid from the punctum into the nose, and more certain in demonstrating no thoroughfare. The fact that there is an overflow of tears will not suffice, because such an inflammation induces lacrimation. If the fluorescin passes into the nose the passages are open and dacryocystitis is excluded, but if it does not the only possible lesion, aside from an acute purulent dacryocystitis, is the rupture of an empyema of the ethmoid or frontal sinus that presses upon the upper part of the passage in such a manner as to occlude it. This is differentiated by an inspection of the seat of the swelling, which is mainly below the internal palpebral ligament in dacryocystitis, but chiefly above it in a ruptured empyema of one of these sinuses.

Rare cases have been demonstrated of abscess of the lacrimal sac with no prior lacrimal disease, but the question whether the sacs were eroded by abscesses in their immediate vicinity, or became infected with virulent microorganisms, perhaps from some other part of the body, has usually been left unanswered. In one such case that followed an attack of typhoid fever I obtained a pure culture of typhoid bacilli from the pus in the sac.

Lacrimal Fistulæ

A small opening in the skin of the cheek, from which trickles or oozes fluid or mucus, is the mouth of a fistula, which leads to some place where fluid is secreted. Whether we are able to obtain a history of lacrimal trouble or not, we wish to learn whether it is connected with the lacrimal sac, and the simplest way to do this is to inject a little fluorescin solution into the canaliculus of one of the

lids on the same side of the face, clear away any dried secretion that may have accumulated about the opening, and observe the color of the fluid which appears a few minutes later. If a drop of stained fluid appears after a few moments the fistula is lacrimal. Such fistulæ may be very tortuous, and their mouths may be far from where we would naturally expect to find them; they may be almost anywhere on the cheek, and as far down as the upper lip. Sometimes they are so very small that the external opening is scarcely visible and makes its presence known only by the occasional formation of a drop on the skin; these are called **capillary fistulæ.**

Dacryoadenitis

When a patient is attacked suddenly with pain in the upper outer part of the orbit, together with tenderness, redness, and swelling of the outer part of the lid, chemosis of the outer part of the bulbar conjunctiva, and an exophthalmos downward and inward, he may have an **abscess** in that part of the orbit, an **orbital cellulitis,** or an **acute dacryoadenitis.** The first two conditions are excluded when a very tender, swollen body can be felt protruding from beneath the upper outer edge of the orbit, and when eversion of the lid brings the swollen gland into view, but such evidence cannot always be obtained. Sometimes the lid cannot be everted, and when suppuration has taken place already, nothing but a fluctuating mass can be felt, so that in some cases the differentiation cannot be made until after the evacuation of the pus. It is probable that the palpebral is affected more often than the orbital portion of the gland, and that an inflammation of the latter is more difficult to distinguish from an orbital cellulitis. Acute dacryoadenitis is quite uncommon. Perhaps it occurs most often in connection with mumps, when it is frequently known as mumps of the lacrimal gland, but it has been observed after quite a number of other acute infectious diseases, during the course of gonorrhea, and it has been ascribed to other infections. It may terminate in resolution, in suppuration, or in a chronic form of inflammation.

A firm, lobulated tumor in the same situation which causes a prominence in that part of the upper lid, renders the outer part of the palpebral fissure abnormally narrow, displaces the eyeball somewhat forward, downward, and inward, may be pressed back more or less beneath the margin of the orbit, and is brought into view be-

neath the conjunctiva on eversion of the upper lid, is an **enlarged lacrimal gland.** If it is tender to the touch, and perhaps is associated with a certain amount of œdema of that part of the lid and of the transitional fold of conjunctiva, the condition is that of chronic dacryoadenitis, otherwise the gland contains a firm tumor or is hypertrophied. Hypertrophy of the lacrimal gland may be congenital or acquired, and sometimes becomes so great as to cause an extreme degree of exophthalmos. In the majority of cases it is quite difficult to determine whether the gland is hypertrophied or the seat of a tumor prior to extirpation, but occasionally we can obtain some symptoms that guide us. If the swelling subsides rapidly under antisyphilitic treatment we may safely conclude it to be of luetic origin. Rapid growth arouses suspicion of a malignant neoplasm. If the growth is rapid, is associated with enlargement of the cervical lymphatic glands, leucocytosis, and hemorrhages from the mucous membranes, and if it is seen to be of a greenish color when the lid is everted, it is a chloroma.

Dacryops

When the upper outer part of an uninflamed upper lid is bulged forward by a painless, fluctuating tumor, and eversion reveals a translucent, bluish or pinkish cyst that enlarges when the patient weeps, it is probable that we have before us a dacryops, or retention cyst of the lacrimal gland, although it is possible for a hydatid cyst to be met with in this situation, and atypical cases are met with at rare intervals. In one that I reported as a cystic tumor of the lacrimal gland in the Archives of Ophthalmology for 1891, the eye was flattened from above downward, and pressed downward and forward so as to cause lagophthalmos, by an insensitive, uninflamed, fluctuating tumor. The bluish appearance of dacryops was not visible when the lid was everted, and the patient said that tears came from the eye when she cried. The tumor was found to be composed of three cysts, a large one extending back an inch and a half from the supraorbital ridge, with its wall firmly adherent to the periosteum, and two small ones, one beneath the angular process of the frontal bone, the other almost wholly in the lid.

A little aperture in the skin of the upper lid from which lacrimal fluid exudes is the mouth of a **fistula,** and we have to learn from the history whether it was congenital, or was caused by a wound,

by the rupture of an abscess of the lacrimal gland, or by an external incision into a dacryops.

THE LYMPHATIC GLANDS

When we first glance over the face we note whether there is any swelling in front of the tragus, where the **preauricular gland** is situated, and this region may be palpated with the finger to learn whether this gland can be felt. If it is enlarged so that we can feel it we note whether it is small or large, hard, elastic, or soft, tender or insensitive, the seat of inflammation or not. Most of the lymph channels from about the eye pass through this gland, which arrests infectious organisms brought in the lymph current, in an attempt to prevent a general infection of the organism, and the nature of the adenopathy, when present, depends to a great degree on their nature, number, and virulence. The gland may be slightly swollen and tender, large and suppurating, or hard and insensitive. As a rule the swelling is not very marked, but it accompanies many acute infections of the lids and conjunctiva. The more severe the parent inflammation the greater usually is the swelling of the gland, so it is apt to be greatest in such infections as anthrax, vaccine ulcers, and hordeola of the lids, and streptococcal diseases of the conjunctiva, but this is by no means always the case. On the contrary it may be absent in a case of violent conjunctivitis of a nature in which it usually is present, like gonorrheal ophthalmia, or present where ordinarily absent, as in phlyctenular conjunctivitis. When the swelling is considerable a similar condition can be detected, as a rule, in the cervical glands, but when it is slight the gland seems to have been able to arrest the organisms.

The adenopathy caused by a ***chancre*** of the lid or conjunctiva is hard, indolent, insensitive, never suppurates, but extends slowly to all of the neighboring glands. That present in ***tuberculosis*** of the lids or conjunctiva may be indolent, but usually ends in suppuration; it seldom occurs in connection with lupus. Small ***ulcers*** of the conjunctiva of indefinite origin may induce a well marked adenopathy. That of ***Parinaud's conjunctivitis*** is strongly marked and lasts for months after the conjunctiva is well; sometimes the glands suppurate, sometimes they do not. The slow, hard swelling associated with ***cancer*** occurs late in the course of the disease, as a rule, and indicates the involvement of the glands. Occasion-

ally an affection of the preauricular gland with no explanatory lesion about the eye suggests that an infection has entered through the conjunctiva without producing trouble there; this has been noticed in glanders.

The **submaxillary lymphatic gland** is apt to be affected in the same manner by lesions situated about the inner canthus, as it is located in the course of a part of the lymphatics that come from the inner portions of the lids and the adjoining tissues.

THE ACCESSORY SINUSES

No account of the symptoms presented by the eye would be complete without mention of those met with in cases of inflammation of the accessory sinuses, although none are of themselves diagnostic. Certain groups render a diagnosis highly probable, but they have to be supplemented by rhinological examinations, transilluminations, roentgenographs, and sometimes exploratory procedures.

A sudden onset of œdema of the upper lid, perhaps with ptosis, accompanied by pain and fever, in a patient who gives a history of severe pain at the root of the nose, or in the brow, on catching cold, is very suggestive of **empyema** of the ***frontal sinus.*** If the œdema is confined mostly to the inner third of the lid, and is associated with severe neuralgic pain, imperfect motility of the eyeball, diplopia and lacrimation, we think of the ***anterior ethmoidal cells*** as the more probable site of the inflammation. If to these symptoms are added an exophthalmos downward and outward, or outward and a little downward, we feel pretty sure that pus is to be found in one of these two localities. But the symptoms may not be so characteristic, and other pathological conditions than pus in these sinuses may give rise to similar ocular symptoms. A slowly progressive exophthalmos downward and outward with no signs of inflammation is very apt to be due to a **tumor** that has distended the walls of the frontal sinus, or has entered the orbit from that cavity, though it may be caused by a mucocele, an encephalocele, or a tumor springing from the tissues in the upper, inner, front part of the orbit. If a hard painless tumor is felt in this situation and the brow is pressed forward, we may have to deal with an ivory osteoma of the sinus that arises at the junction of the frontal and

ethmoid bones, distends the walls of the sinus in every direction, and sends offshoots into the neighboring cavities, including the orbit. A similarly progressive exophthalmos outward and a little downward suggests a mucocele or tumor of the anterior ethmoidal cells.

When a patient has a persistent œdema of the lower lid associated with a swelling of the cheek that is not especially marked along the side of the nose, and pain in the side of the face, either constant or periodic, we think of an **empyema** of the ***antrum.*** Our suspicions are strengthened if there is a chemosis of the conjunctiva, dilatation of the retinal veins with some œdema of the papilla, and still more if to these symptoms is added an exophthalmos upward, or upward and outward. Such an exophthalmos without an inflammatory œdema of the lid might indicate a new growth in the antrum.

An **orbital cellulitis** of unknown origin always demands a thorough investigation of the accessory sinuses, for purulent sinusitis is a very common cause. An orbital cellulitis may be set up very quickly by a sinusitis when the agents are extremely virulent, but ordinarily the history will indicate that the sinusitis is not recent, rather that it has existed a long time. If the inflammation exists anteriorly the patient probably has had symptoms which were referable to the frontal sinus, the anterior ethmoidal cells, or the antrum of Highmore; if it is in the posterior sinuses he may have an impairment of vision with fundus changes that are insufficient to explain it, or unaccountable themselves. We may find a tender point within the orbit from which the globe is displaced when the inflammation is anterior, or a direct protrusion of the eyeball when it is posterior; the former when the infection of the retrobulbar tissue is preceded by an osteitis and periostitis of the wall of the orbit between it and the sinus, the latter when such an osteitis is too far back to allow the swelling of a periostitis to impinge on the surface of the eyeball, and the globe itself is crowded forward by the displacement of the retrobulbar tissues.

DeSchweinitz has called attention to a recurrent or fugitive œdema of the lids, with or without reddening of the skin, that may vary from a slight to a marked swelling, and comes and goes at intervals, the attacks associated with intense headache, while there may be no trouble or discomfort during the intervals, and to a fugitive ecchymosis of the lids, also attended by pain during the

attacks, both of which he has found in connection with inflammation in the frontal sinus and the anterior ethmoidal cells. Probably in these cases the pus drains away through the natural channels, and the attacks are caused by temporary occlusions of the latter. The same surgeon has observed a fugitive episcleral congestion to be sometimes indicative of sinus trouble, and has seen a distinct œdema of the corneal epithelium in a few of the more violent cases.

A most serious condition confronts us when a patient complains of a rapid deterioration of the vision of one eye for which we can discover no adequate reason, and this one symptom is enough to call for a rigid and thorough investigation of the **posterior ethmoidal cells** and of the **sphenoidal sinus.** It is not enough that no pus can be seen to exude from the openings of these cavities, or that a roentgenograph is negative, these cavities should be skillfully catheterized and the absence of pus demonstrated beyond a doubt, for not only the sight of the eye, but the life of the patient may be threatened. We learn little from an ophthalmoscopic examination in these cases. Rarely the retinal vessels are occluded; more often we find a papilloedema, an optic neuritis, or a neuroretinitis; still more often we can make out nothing more than a slight blurring of the margins of the disk, or a diminution in the size of the retinal vessels, and in many cases even these slight signs are wanting. We gain more from a study of the field of vision, particularly if we find an enlarged blind spot, or a scotoma, for either of these may be accounted for by a posterior sinusitis. Such a scotoma usually is central, may be relative for color at first, later absolute for color and relative for white, and may be of any shape, round, oval, or triangular, but it may be paracentral, ring shaped, or hemianopic. In other words, a scotoma exactly like one found in retinal troubles, lesions of the chiasm, and toxic amblyopia, sometimes occurs in connection with empyema of the sphenoidal sinus. An additional symptom sometimes present is that movements of the eyeball cause pain. When a thorough examination of the accessory sinuses has demonstrated the absence of inflammation in any one of them, we must bear in mind that an abscess in the zygomatic fossa is a possible cause, though probably it is such only in very rare cases.

CHAPTER IV

Displacements of the Eyeball

Next in order we observe whether the eyeballs occupy their normal positions in the orbits and maintain their proper relations to each other. An eyeball which occupies an abnormal position as a whole in the orbit is displaced; when the visual axis of one is not parallel with that of the other the eye is said to deviate. Displacement of an eye is almost invariably associated with deviation, but deviation often is met with without displacement, so the two will be considered separately. One orbit may be placed higher than the other and produce a vertical displacement of the eyes with reference to each other, but this is not included because each globe occupies its normal position in its own orbit. At the same time such a condition as this must not escape notice, as it is a serious cause of eyestrain.

ENOPHTHALMOS

A sunken eye, its upper lid drooping from lack of support, is in the condition of enophthalmos. The eyes recede a little when the lids are closed tightly, or are pressed upon, but this physiological recession is not included. If we see no eye when we lift a drooping lid, we have to learn from the history whether it was absent at birth, or has been enucleated, though the latter may be indicated by the presence of scars. If the eye is abnormally small, with no signs of injury or disease, and the condition is congenital, the case is one of microphthalmos. Should there be a history of inflammation or injury of a previously good eye, and the globe be both small and deformed, it is atrophic. It is only when the eyeball appears to be of normal size that we speak of enophthalmos, and then we have to inquire into the cause.

Bilateral enophthalmos appears in atrophy of the orbital fat, whether caused by disease or old age; in these cases the condition and the cause are recognized readily.

A slight enophthalmos of one eye sometimes is seen during an

attack of migraine, and may be caused in very rare cases by a spasm of all four recti. *Ball* says that three cases have been reported in which the eye receded when the lids were separated forcibly and returned to place when the force was removed.

When a patient presents a slight degree of enophthalmos of one eye with a myosis that is not affected by cocaine, and redness and warmness, or perhaps abnormal paleness of the same side of the face, some lesion implicates the cervical sympathetic on the same side. This may be a swelling of the thyroid, a tumor in the neck, posterior mediastinum, or upper part of the chest, an aneurysm of the carotid, an injury of the neck, an excision of the superior cervical ganglion, a traumatic paresis of the brachial plexus, or syringomyelia.

A *very great enophthalmos of one eye* is nearly certain to be associated with scars about the orbit, for almost always the cause is a violent traumatism. The patient presents a deeply retracted eye over which the lids are sunken, and the palpebral fissure narrowed, so that he looks much as though he were wearing an artificial eye. The displacement may be directly backward, or downward with a deviation upward. The mobility is much impaired. The vision may be lost, or it may be good, and in the latter case the patient has diplopia, at least when the upper lid is lifted. Quite a number of theories have been advanced to account for this displacement when it occurs as the result of a direct traumatism, but no one is universally satisfactory, so it is probable that the lesions which cause the eyeball to recede are not the same in all cases. The history of the removal of a tumor from the orbit furnishes a sufficient explanation, and so does one of an orbital cellulitis, as this may leave cicatricial bands that draw the eye back by their contraction.

Retraction of the eyeball into the orbit during the movements of the eye is one of the symptoms of a rare syndrome caused by a congenital deficiency of certain extrinsic muscles, especially the externus, or by tense cicatrices that furnish an unyielding resistance to the endeavors of other muscles to turn the eye in the opposite direction.

EXOPHTHALMOS

The essential feature of exophthalmos is that the entire eyeball is projected forward in the orbit. When the anterior portion projects while the eyeball as a whole maintains its normal position, as

in infantile glaucoma, in some cases of myopia, and when the margins of the orbit are developed imperfectly, a fault that is particularly noticeable when it is confined to one side of the face, the prominence of the eyeball does not constitute exophthalmos. A slight amount of movement forward on the part of the eyeball seems to be ***physiological,*** for *Birch-Hirschfeld* has proved by photographs that our eyeballs protrude a little when we stoop, and when we open our lids widely, but this again is not meant when we speak of exophthalmos.

A displacement forward is produced ***mechanically*** by a relaxation of the tissues that hold the eye back, or by a reduction of space within the orbit through an increase of its contents, or an encroachment upon this space by the bony walls. The physiological protrusion of the eyes may be increased by the congestion caused by compression of the facial veins, and such a congestion may produce a distinct exophthalmos when the jugular veins are compressed in the neck. The increase of the orbital fat sometimes makes the eyes of corpulent persons very prominent, but unless the prominence is considerable it is of little consequence. Exophthalmos is congenital in certain deformities of the skull in which the orbit is shortened and the eyeball pushed forward; in these cases it varies in degree according to the amount of deformity of the orbit, it may be slight or very great, but it is irreducible and not likely to be progressive.

The eyeball may be displaced not only forward, but also upward, downward, or in any lateral direction, according to the location of the force which displaces the contents of the orbit. It is important to note the extent and direction of the displacement, whether it is reducible or not, whether its progress has been rapid or slow, the motility of the affected eyeball and of the lids, and the presence or absence of any signs of inflammation.

A slight protrusion can be detected when the extrinsic muscles of the eye are paralyzed, and is more apparent when the affection is confined to one eye alone, but it is very subordinate to the other symptoms and adds nothing to help us in diagnosis. It always is slight and is never productive of serious consequences. A similar slight protrusion often is to be seen after a free tenotomy of one of the muscles, especially of the internal rectus of one eye, when the history and perhaps a scar in the conjunctiva is likely to furnish the explanation.

Well marked exophthalmos usually indicates a serious condition. It may be produced by certain affections of the nervous system, usually is present in exophthalmic goiter, and above all is the cardinal symptom of nearly all of the diseases that occur in the orbit. We note the condition of the surrounding tissues, any general symptoms that may be present, the presence or absence of any swelling or sign of inflammation in the lids or face, introduce the tip of a finger within the margin of the orbit and feel about for any point of abnormal resistance or tenderness, investigate the condition of the nose, transilluminate the accessory sinuses, and have roentgenographs taken. Any or all of these measures may be necessary to determine the diagnosis in any particular case, but sometimes the associated symptoms suffice to direct our attention to a specific cause.

A **slight** exophthalmos associated with dilatation of the pupil and increased perspiration and pallor of the same side of the face, indicates that some lesion is irritating the sympathetic nerve on that side.

Exophthalmic Goiter

The combination of exophthalmos, usually bilateral, widening of the palpebral fissure, decreased frequency in winking, a retarded movement of the upper lid when it should accompany the eye in its downward movement, and a peculiar tremor of the lids when gently closed, with tachycardia, enlargement and palpitation of the heart, a feeling of oppression, pulsation of the large arteries in the neck, sometimes a rhythmic concussion in the head, a swelling of the thyroid, over which a thrill may be felt and a roar heard, and a tremor of the hands when they are held out with their palms down, forms the clinical picture of exophthalmic goiter, or Graves's disease. Other symptoms that may be mentioned are nervousness, mental depression, apprehension, excessive sweating, emaciation, weakness and pain in the limbs, brittleness of the nails, partial alopecia including the brows and lashes, lacrimation, conjunctivitis, and insufficiency of convergence. The tachycardia, exophthalmos, and enlarged thyroid are spoken of usually as the three cardinal symptoms, but the only one of them that can be said to be present in all cases is the tachycardia, and it is a curious commentary on our nomenclature that exophthalmic goiter may exist without exophthalmos, without goiter, or without either. We feel almost

compelled to call it Graves's disease, after the Englishman who first described it as an individual affection, as is done in Great Britain. Germans call it Basedow's disease from the name of their countryman who amplified *Graves's* description some years later.

Hemorrhage into the Orbit

Sometimes an exophthalmos appears suddenly together with ecchymoses in the lids and beneath the bulbar conjunctiva, and we meet with a firm resistance when we try to press the eyeball back. A hemorrhage has taken place behind the eyeball, usually as the result of traumatism. If a severe contusion has fallen upon the face in the vicinity of the eye, we conclude that there is a **fracture of the wall** of the orbit, or that blood vessels within it have been lacerated. A penetrating wound may produce the same result. The amount of exophthalmos, the direction in which the eyeball is displaced, and the degree to which its movements are restricted, vary in accordance with the position and extent of the hemorrhage. When the contusion fell upon some part of the cranium we infer from these symptoms that it produced a fracture of the base of the skull. The same local condition is occasionally produced **without traumatism,** in persons who have brittle blood vessels, by a rupture of the orbital vessels during straining, stooping, or coughing, and a few cases are on record in which even such a slight actuating cause was denied. The hemorrhage may be altogether out of proportion to the traumatism when the patient is hæmophilic. The consequences to the eye of a retrobulbar hemorrhage may be serious through the pressure exerted on the optic nerve, the interference with nutrition, and the exposure of the cornea to the air when the exophthalmos is so great that the lids cannot close over it, but an immediate blindness tells us almost with certainty that the optic nerve itself has been wounded.

Emphysema of the Orbit

Sometimes a rather similar picture is seen after a contusion in the region of the orbit in which the lids, aside perhaps from some ecchymoses, are pale, swollen, and closed over the exophthalmic eyeball, which may be pressed back somewhat into the orbit. Percussion over the globe elicits a tympanitic note. This is an emphysema of the orbit. It indicates that an opening has been made in

the bony wall which communicates with one of the adjacent air chambers and permits the entrance of **air.** We do not obtain the feeling of crepitation under our fingers that we do in emphysema of the lids, unless the air has entered both the orbit and the lids, as is frequently the case.

Luxation of the Eye

When an eye has been luxated by the introduction of a foreign body into the orbit the cause is evident, as a rule. Occasionally such a mutilation is self-inflicted in insanity by passing the finger deep into the orbit.

There is little chance for error in any of the above mentioned conditions, but in others the keenest powers of observation and deduction may try in vain to determine the exact cause of an exophthalmos prior to operation. In the majority of cases in which a protrusion of the eyeball has developed as it were spontaneously it has been caused by a tumor, yet sometimes we have to deal instead with a syphilitic or tuberculous disease. When a tumor is present it is well for both the patient and the surgeon if we can determine before operation whether it is malignant or benign, and if benign whether it is vascular, cystic, bony, or fibrous, whether it is fixed or freely movable, and where it is situated, for all of these things bear upon the method of intervention to be chosen. We should feel humiliated if, after performing Krönlein's operation, we found that the tumor was on the opposite side of the orbit, was one that could have been removed through an incision in the conjunctiva, or was an ivory exostosis from the frontal sinus which must be approached in a different way, and it is only less mortifying to ascertain, after we have entered upon some other form of operation, that we should have recognized that a resection of the outer part of the wall of the orbit was necessary.

Exophthalmos Due to Syphilis and Tuberculosis

If a patient with exophthalmos of unknown origin gives a history, or presents clinical signs of syphilis, a Wassermann or a Noguchi test should be made, and an energetic antiluetic treatment tried. *Neither a positive Wassermann, nor indubitable evidence of active syphilis, suffices* for a diagnosis of syphilitic disease of the orbit, for any kind of a tumor may develop here in a syphilitic as

well as in anyone else. The only positive proof is the rapid subsidence of the exophthalmos under antisyphilitic treatment.

Tuberculosis of the retrobulbar tissues is met with very rarely, and then in elderly people. Positive differentiation from a tumor is not yet possible, but the probability of such a diagnosis is enhanced by the presence of tuberculous lesions in or about the eye, as well as by a local reaction to the tuberculin test.

Exophthalmos Caused by Tumors

We need to learn from the ***history*** the nature of the first signs of trouble and when they were first noticed. Often the protrusion of the eye is the first thing observed, but sometimes this is preceded by a persistent headache, perhaps followed by a deterioration of vision. We ascertain whether there was an antecedent traumatism, and whether the patient is suffering from some disease, like leucocythæmia, in which the development of orbital tumors is symptomatic, or, if the appearance of the patient is such as to suggest this, we have an examination made of the blood.

We notice whether the eyeball projects axially from the orbit, or is displaced in any other ***direction,*** and we measure this displacement, as it may give us a clue to the size and location of the tumor. Then we introduce the tip of a finger as deeply as possible within the margin of the orbit, starting at the place indicated as the probable site by the inclination of the eyeball, and feel for an object that presents an abnormal resistance. If we find such an object we try to determine its consistency, whether it is hard, firm, or soft, to ascertain whether it is fixed or movable, situated deeply or superficially, and, so far as we may, its size and structure. We learn much that is of service in this way. To take a rare but very plain example, if the exophthalmos has developed very slowly, was preceded for a long time by persistent frontal headache, if the brow is protuberant and has slowly grown more so along with the exophthalmos, if the eyeball is displaced forward, downward, and outward, and if the finger beneath the upper, or upper and inner portion of the margin of the orbit impinges on a hard, immovable object, it is almost certain that the patient has an ivory osteoma growing from the junction of the ethmoid and frontal bones, which distends the frontal sinus and pushes its processes toward or into the neighboring cavities of the orbit, the nose and the cranium.

Very slow ***growth*** increases the probability that the tumor is benign; very rapid growth strongly indicates that it is malignant. The history of antecedent symptoms referable to the development of a tumor in one of the accessory sinuses, the nose, or the adjacent part of the cranium, render it likely that the orbit is involved secondarily. If the eyeball is displaced downward the tumor probably is above, though not necessarily so, for if it happens to be situated on the floor of the orbit in such a place that it presses the posterior end of the eyeball upward, the anterior end will be tilted down so as to produce the same picture. The tip of the finger can be introduced quite a distance into the orbit, carrying the lid before it, especially when the eyeball has been pushed aside, and the feeling when it impinges on a hard, immovable substance is unmistakable. The same sense of touch tells us when the body feels tense and firm, or soft and fluctuating. By pressing upon it we learn whether it is movable, or fixed to the periosteum. If we can move it about we conclude that it originated from the soft tissues in the orbit; if it is movable with the eyeball we know that the two are intimately connected. If fluctuation can be detected, it is a rather superficial cyst which may contain serum, lacrimal fluid, echinococcal fluid, cerebrospinal fluid, or blood encapsulated after a hemorrhage. If it is in the upper inner angle of the orbit it may be an encephalocele, or a meningocele at the junction of the frontal, superior maxillary, ethmoid and lacrimal bones, or a distended mucous bursa of the trochlea; the herniæ cerebri are congenital, the distended bursa usually is acquired. The differentiation can be made in all of these conditions by aspiration and examination of the fluid. It is unnecessary to give in detail the chemical and cytological properties by which we distinguish serum, blood, lacrimal fluid, and cerebrospinal fluid, but it may be well to say that when we find a fluid that has a high specific gravity and contains hydrochloric and succinic acids, the cyst is a hydatid, which usually is attended by pain and inflammation.

Cysts that are situated so ***deeply*** in the orbit as to be beyond the reach of the exploring finger are apt to produce a nearly or quite axial exophthalmos. The presence of a blood cyst may be inferred rather than diagnosed from a history of a retrobulbar hemorrhage in the distant past, after which the exophthalmos decreased somewhat, but never passed away entirely. An encephalocele or meningocele may protrude through the superior

orbital fissure or the optic foramen, and as long as such a cyst, which is congenital, remains in open communication with the brain cavity, the pulsation of the brain will be communicated to it and may be transmitted by it to the exophthalmic eyeball. The differentiation of this condition will be considered under pulsating exophthalmos.

A firm, slowly growing tumor in the upper outer angle of the orbit may be a swollen lacrimal gland, or a **dermoid.** This is the favorite situation of the latter, though it may occur elsewhere. If the mass can be pressed back beneath the margin of the orbit it is the gland, perhaps containing a tumor, but if it cannot the probability is that it is a dermoid. In the latter case we try to ascertain by manipulation whether it extends far back into the orbit and will require a Krönlein operation for its removal, or can be shelled out like the lacrimal gland through an incision in the conjunctiva.

A firm, freely movable tumor, that has grown very slowly, situated anywhere between the eyeball and the wall of the orbit, is either a benign neoplasm or a fibrosarcoma; the differentiation cannot be made clinically. An optic nerve with its sheath distended with fluid may bend upon itself and form a knuckle which may give the same sensations to the exploring finger as a fibroma from the muscular or connective tissue, as well as displace the eyeball in the same manner. Such a case forms an exception to the rule that *tumors of the optic nerve displace the eyeball* nearly or quite *axially,* which is one of the chief diagnostic features of these growths. When exophthalmos appears early, increases slowly without pain, and is directly forward, we believe it to be caused by a tumor of the optic nerve, which usually is benign. An additional symptom is that, as a rule, the eye becomes blind at an early age, with symptoms of optic neuritis followed by atrophy.

Exophthalmos may affect either one or both eyes in acromegaly; it is uncertain whether it is caused by a proliferation of fat in the orbit, or by compression of the cavernous sinus. It is seen very rarely in hydrophthalmos. If the exophthalmos is bilateral, and tumors can be felt in both orbits, we must think of lymphomata, or of Mikulicz's disease.

A rapidly growing tumor of the orbit probably is malignant. A **carcinoma** is seen very seldom and usually starts in the neighboring tissues, the lids, the lacrimal gland, or the accessory sinuses,

especially the antrum. It can be diagnosed clinically only from the presence of carcinoma elsewhere. **Sarcoma,** on the contrary, is quite common, starts from every possible tissue in the orbit, and appears at any age. All forms are met with, ranging from the rapidly growing and very malignant small cell variety to the slowly growing fibrosarcoma, which is hard to distinguish clinically from a benign tumor for a long period in its course. Pigmentation usually is absent. When tumors have grown very rapidly in both orbits we may suspect the rare and malignant chlorosarcoma or chloroma, and this suspicion becomes certainty if they have a greenish hue when seen through the conjunctiva.

It seems almost incredible that a considerable degree of exophthalmos can develop and persist, and yet nothing be found to explain it after an exploratory operation. Such an occurrence might be referred to the unwitting evacuation of a cyst if the exploration were made through the conjunctiva, or by means of an enucleation of the eyeball, but *Roemer* pictures in his textbook a case in which the eye was quite protuberant and dislocated downward, but no tumor was found after he had performed Krönlein's operation. In a similar case of rapidly advancing axial exophthalmos in a little child I resected the outer wall of the orbit, but could find nothing except an œdematous condition of all of the tissues. Microscopical examination of these tissues proved it to be a case of

Pulsating Exopnthalmos

When a patient presents a red, œdematous upper lid, its skin full of dusky, dilated veins, hanging tensely down over an exophthalmic eyeball, the conjunctiva of which is chemotic and shows many dilated veins, if we can feel a rough thrill and pulsation when we place a hand on the protruding eye, and can hear with the stethoscope a blowing murmur that is audible to the patient and is the source of much discomfort, we say that he has a pulsating exophthalmos. The vision may not be affected, or it may be destroyed as the result of an ischæmia of the retina, caused by the compression of the central artery, and the consequent atrophy, or of a venous engorgement of the papilla and retina, which is often accompanied by retinal hemorrhages. The eye pulsates synchronously with the heart. Such a pulsating exophthalmos as this is due to an **arteriovenous aneurysm of the carotid and the cavernous sinus, or an aneurysm of the carotid within the sinus,**

which may be caused in two ways—with and without traumatism. As a rule, it is caused by a fracture of the skull, occasionally by a gunshot wound, and considerable time may elapse, perhaps months, after the injury before the picture develops. Less often a person who is stooping or coughing notices a cracking sound, feels a severe pain, and has a pulsating exophthalmos appear quickly. This has been observed in elderly people and in pregnant women, but can happen only when the wall of the carotid is not normal. In both of these varieties the eyeball recedes when the carotid of the same side is compressed, the movements of the globe are more or less limited, and frequently one or more of the muscles are paralyzed, giving rise to a deviation of the eye.

A pulsating exophthalmos in which there are few, if any, signs of venous engorgement in the lids or conjunctiva, no thrill is felt, and no murmur heard through the globe, may be due to a **meningocele** or encephalocele in the back part of the orbit which is in open communication with the brain, to a **vascular tumor** that receives pulsations from some artery, like an aneurysm of the ophthalmic artery, or to a **solid tumor** so placed as to receive and transmit arterial pulsation. If the condition is congenital and there has been no traumatism, the trouble probably is a hernia of the brain, either a meningocele or an encephalocele, into the back part of the orbit, and this is to be thought of first when the patient is a child. When the condition has been acquired by an older patient, we have to deal with either a solid or a vascular tumor, and to differentiate between these we inquire into a possible history of traumatism, which may have been of such a nature as to injure the retrobulbar blood vessels, and investigate the vascular system for any disease that might explain the formation of an aneurysm. In the absence of these we may conclude that a solid tumor is present, but we cannot be positive in all cases.

Exophthalmos Caused by Vascular Trouble

Other vascular troubles may cause an exophthalmos that does not pulsate. An angioma may start in the lid and extend slowly into the orbit until the eye is made to protrude, or one may form deep in the orbit, frequently in the muscle funnel, where it often is congenital. The former is not hard to recognize, but the latter may be very difficult. The exophthalmos varies in degree from time to time and is increased by any strong emotion, or by stoop-

ing, unless the angioma happens to be encapsulated, so the presence of this symptom leads us to suspect angioma at once. The finger palpating between the eyeball and the orbit may fail to detect any growth, or it may impinge on a soft, cushiony, elastic mass that does not pulsate. If the tumor has extended forward far enough to be visible externally, it forms a doughy, purplish swelling that partially closes the lids over the eye. The exophthalmos is not great, and is not wholly reducible by pressure, though in all vascular tumors the eyeball can be pressed back more or less into the orbit. If the exophthalmos is bilateral we should look for an aneurysm of the basilar artery.

Very rarely we meet with a patient in whom one eyeball protrudes unduly when he stoops, and returns to its normal position as soon as he holds his head erect. This is supposed to be due to a varicose dilatation of the veins of the orbit and is called an **intermittent exophthalmos.**

Inflammatory Exophthalmos

A sudden protrusion of the eye with pain, fever, swelling and redness of the upper lid, may be a symptom of such widely different conditions as a thrombosis of the retrobulbar veins and of the cavernous sinus, periostitis of the walls of the orbit, inflammation of one or more of the accessory sinuses, orbital cellulitis, tenonitis, and panophthalmitis.

Orbital Cellulitis

When a patient is prostrated suddenly with fever, the eyeball projects directly forward in the axis of the orbit and is more or less immobile, the lids are swollen and the upper one hangs down over the eyeball, the conjunctiva is chemotic, and the dull pain is increased by attempts to move the eye, as well as by pressure upon it, but no points of marked tenderness can be found within the margin of the orbit, and the refractive media of the eye are clear, he has an orbital cellulitis. Its cause may have been a slight, or a severe traumatism that has lodged a foreign body in the orbit. Quite a number of cases are known in which a child has fallen on a sharp stick, or a pointed pencil, in such a way that a portion of the stick was driven through the conjunctiva into the orbit and broken off, where it was hidden and left no

visible wound or other traces, but soon caused a fatal cellulitis unless it was found and removed. Cellulitis may be caused by mumps, tonsillitis, puerperal fever, and other infectious diseases, by a purulent dacryocystitis, a thrombophlebitis of the facial veins, or a panophthalmitis, but in sixty per cent. or more of the cases it originates from an inflammation of one of the **accessory sinuses,** and then the direction in which the eyeball is displaced is of much help in determining which sinus or sinuses contains the primary disease.

Exophthalmos has been observed in rare cases of actinomycosis, glanders and anthrax of the orbit. If an acute exophthalmos appears after a penetrating wound of the orbit, associated with stiffness of the neck, convulsions, facial palsy, and spasms of the muscles of the throat, we know the cause to be tetanus. An acute exophthalmos may be symptomatic of an abscess in or about the lacrimal gland, or of an acute dacryoadenitis, the diagnosis of which has been considered under the lacrimal organs.

Orbital Periostitis

An acute exophthalmos, usually with a lateral displacement, associated with fever, pain, perhaps nausea and vomiting, diplopia, inability to move the eye in certain directions, and a firm or fluctuating, very tender, node on the wall of the orbit beneath a circumscribed redness and swelling of the lid, is indicative of a periostitis at the point of tenderness. Such a periostitis may develop during or after an attack of some **infectious disease,** such as measles, scarlet fever, typhoid fever, influenza, or tonsillitis, or it may be caused by **traumatism.** An **abscess** of the retrobulbar tissue, or an abscess in the zygomatic fossa, must be taken into account after all other causes have been excluded. In the great majority of cases we have to consider whether it is due to syphilis, tuberculosis, or an empyema in one of the accessory sinuses.

A periostitis of the margin of the orbit does not produce exophthalmos unless it extends back of the tarso-orbital fascia, but it calls for a thorough investigation of the organism. If it is situated on the upper margin the chances are that it is syphilitic; if it is on the lower or outer, it probably is tuberculous. Both syphilitic and tuberculous periostitis of the orbit are rare, but both may extend back of the tarsoorbital fascia, and then exoph-

thalmos is added to the symptoms. **Syphilitic** periostitis may appear anywhere on the walls of the orbit, both in adults who have acquired the disease, and in children in whom it is hereditary, so it is well to try the effect of an energetic antisyphilitic treatment when a patient who gives a history or clinical signs of syphilis has a periostitis of the orbit; if the symptoms abate immediately the diagnosis may be considered settled. A **tuberculous** periostitis usually appears in children or young people, frequently after a contusion, and causes exophthalmos very rarely. Its clinical picture differs materially from that of tuberculosis of the retrobulbar tissue, which likewise is rare, but appears in elderly people, develops slowly, and is hard to differentiate from a retrobulbar tumor. In most cases of exophthalmos due to a periostitis we find the cause of the latter to be in one of the **accessory sinuses.** If the tender point is just within the upper inner angle of the orbit, an empyema of the frontal sinus is suggested, if it is over the lacrimal bone we think of the ethmoid, if it is within the lower margin the maxillary sinus probably is the seat of the trouble. A catarrhal inflammation in these sinuses, or a mucocele, rarely excites inflammation in the orbit, while empyemata do so frequently.

A **mucocele** bulges out the wall of the sinus in the direction of least resistance, encroaches on the space of the orbit, and produces a slowly developing exophthalmos in the same direction as when a periostitis is formed, but the inflammatory symptoms are slight or absent. A mucocele may be difficult to differentiate because no pus is to be found in the nose, and its contents do not present the same obstacle as pus to the passage of light, or of the X-rays. Usually the only ocular symptoms are a noninflammatory exophthalmos with its attendant diplopia, but these are absent in mucocele of the sphenoidal sinus.

The exophthalmos induced by disease of the frontal sinus is forward, downward, and outward; that occasioned by an ethmoiditis is forward, outward, and slightly downward; that produced by an inflammation of the antrum is forward and upward. The direction of the exophthalmos together with the locality of a tender point or swelling at the places above indicated, is an excellent guide to the site of the primary inflammation, but alone is not sufficient for a diagnosis; the nose must be inspected, the sinuses transilluminated, and roentgenographs taken, for these may exclude the sinuses even when all the other symptoms point in their direction.

In a case of abscess in the zygomatic fossa these symptoms may lead us to believe that we have to deal with an empyema of the antrum or of the ethmoid when these cavities are free from disease. The same may be true of an orbital cellulitis that starts from the sphenoidal sinus, or the posterior ethmoidal cells, in which the only distinctive point is the early and grave involvement of the optic nerve; but as this may happen in a cellulitis from other causes our best reliance from which to learn the primary seat of the trouble is the roentgenograph.

Diseases of the **posterior ethmoidal cells** and **sphenoidal sinus** rarely cause external ocular symptoms, but are apt to manifest themselves by visual disturbances. Sometimes an optic neuritis, or an occlusion of the vessels, may be visible with the ophthalmoscope, but often the fundus appears to be perfectly normal. Yet even in these cases we may be able to find a central scotoma for color, and cases are on record in which the finding of such a scotoma proved to be the first step toward a correct diagnosis. To what an extent a periostitis is responsible for these symptoms we do not know, but when the retrobulbar space is greatly encroached upon the exophthalmos usually is directly forward.

Exophthalmos Due to Tenonitis

A rather slight, acute exophthalmos, with more or less prostration and fever, an eyeball exquisitely tender to the touch and very painful whenever an attempt is made to move it, and a pale yellow chemosis, are the characteristic signs of a tenonitis, which may be either serous or purulent. In the serous form we usually are able to obtain a history of rheumatism, and the diagnosis is made certain if the symptoms subside in a few days under antirheumatic treatment. The symptoms are more violent in the purulent form, which may be caused by septic infection after an operation like one for strabismus, or of a wound that has opened Tenon's capsule, but quite often it follows influenza. It may cause an optic neuritis, or a purulent iridocyclitis, or the latter disease may give rise to a purulent tenonitis.

Exophthalmos Caused by Cerebral Disturbances

An exophthalmos associated with the serious cerebral disturbances of **sinus thrombosis** is seldom seen, but when it occurs it

usually indicates a septic thrombosis of the cavernous sinus. When the thrombosis is primarily of otitic origin an œdema at the posterior margin of the mastoid aids in the diagnosis. Other ocular symptoms met with in thrombosis of the cavernous sinus are choked disk, optic neuritis, and pareses of the extrinsic muscles of the eye. Conjugate deviations are rare. Thrombosis of the longitudinal sinus sometimes occurs after debilitating diseases, but this produces exophthalmos only when the thrombus has extended to the cavernous sinus; then we may have conjugate deviations of the eyes, or spasmodic movements of them caused by the irritation of the cerebral cortex. The cavernous sinus is the one most apt to become thrombosed after erysipelas, and after diseases of the frontal and sphenoidal sinuses, so it is of practical importance for us to know that when the exophthalmos followed the grave cerebral symptoms the thrombosis is extending from within outward, while if the orbital symptoms appeared first a thrombophlebitis has spread from the orbit to the cavernous sinus, for such a thrombus may originate from an orbital cellulitis.

CHAPTER V

Deviations of the Eyeball

An eye is said to deviate when it does not maintain its normal position relative to the other. When we speak of the normal relative positions of the two eyes we mean that as long as the person is awake and conscious, their visual axes are parallel while the organs are at rest, *i.e.,* looking into infinite space, and converge equally when looking at a finite object, though we cannot say that the upward and rarely parallel position assumed during sleep or unconsciousness is not just as normal. During waking hours this position is maintained by the balance of the tension of the extrinsic muscles, which act in unison in all movements of the eyes under the guidance of nervous impulses which start from various centers and are transmitted along certain nerve tracts. These muscles, nerve tracts and centers constitute the **motor mechanism** of the eyes, part of which is well understood, but we have as yet a very imperfect knowledge of many of its extremely important nerve centers and tracts. We know that when all of the parts of this complex mechanism work in harmony the normal relative positions of the eyes are maintained in a perfect balance, and that a fault at any point induces an abnormality in the ocular movements. Sometimes we can locate such a fault, and perhaps determine its nature, but in many cases we cannot.

This mechanism seems to be incomplete at birth, for babies move their eyes about with a cheerful disregard of their normal relative positions, although the latter are assumed and maintained as the children grow older. Consequently we need not worry over the various deviations to be observed in a baby's eyes unless an abnormal position is taken and maintained for some time. In such a case we may determine whether any of the muscles are paralyzed or not by the test suggested by *Bartels.* Rotate the body of the child to the right; normally the eyes remain directed to the left at first and then turn rapidly to the right; turn the body again to the left when the rotation of the eyes has stopped. If the

movement of either eye is imperfect as the body is rotated, the muscle which should have acted is paralyzed.

In an older child, or an adult, a deviation of an eye may be due to strabismus, or to a paresis of one or more muscles. There is a fault in the motor mechanism in both conditions, but in the latter it is a morbid lesion, while in the former it probably is developmental. The differentiation between muscular paresis and strabismus is to be made first of all, for in the first the deviation of the eye is a symptom of some disease which needs treatment, while in the second we have to deal with the condition itself. In some cases it is easy to make this differentiation, but in others it is quite the contrary.

DIFFERENTIATION OF STRABISMUS AND MUSCULAR PARESIS

We note the age of the patient, and inquire when the deviation was first noticed. Muscular paresis may occur at any age, but a convergent strabismus usually appears between the ***third*** *and* ***fifth*** years, often is said to have followed an attack of measles, whooping cough, or some other sickness, and never develops after binocular single vision has been established unless the sight of the deviating eye has been lost, or the eye has been drawn in by a cicatrix. Divergent strabismus usually appears about the age of ***ten*** or ***twelve.*** In children less than three years old a constant deviation is most likely to be paretic, an alternating or intermittent one to be strabismic, while irregular deviations which are neither constant, alternating, nor intermittent, are of no importance except as showing that the mechanism of the nervous control of the eyes is not yet perfect. In a child four years old and upward the chances are that an inward deviation is strabismic, and it certainly is so if it is alternating or intermittent, but as the age is no protection against the formation of lesions which impair the motor mechanism, tests must be made to exclude paresis of the muscles. The sudden or gradual appearance of a deviation in an adult who has enjoyed binocular single vision must be due to loss of vision in the affected eye, the contraction of a cicatrix, or muscular paresis.

We need to be acquainted with a number of tests, for some that are well suited to intelligent adults cannot be used satisfac-

torily with children, or when intelligence is lacking, and a paresis may vary from a slight disability, which is noticeable only when the patient looks toward a certain part of the field, to a total paralysis of the affected muscle or muscles. When the deviation is well marked the simplest test is to have the patient look at some object, like the examiner's finger, nose, or eye, measure the degree to which the affected eye deviates, cover the good eye with a hand or screen, and measure the deviation that has taken place in it. When we cover the good eye so that it can no longer see the object fixed upon the deviating eye will turn so as to fix the object instead, provided its vision is good enough to enable it to see the object, and the good eye will deviate. We call the original deviation of the affected eye the **primary,** that of the good one behind the screen the **secondary.** This test gives us two valuable diagnostic signs. If the secondary deviation is exactly equal to the primary, the case is one of strabismus; if it is greater than the primary we have to deal with muscular paresis. If the movements of the two eyes to fix are equal as each is covered alternately, the deviation is strabismic; if the movement of one is greater than that of the other, it is paretic. The commonly accepted explanation of these facts is that the innervation necessary to cause either eye to fix is the same in strabismus, while a much stronger innervation has to be sent to a paretic muscle to cause it to act than is needed by a normal one, and as the coacting muscle in the other eye receives the same amount of innervation it contracts excessively and thus produces a greater degree of secondary deviation. As the eye is drawn farther aside by a greater secondary deviation its movement to fix must be greater than that of the other. This test is applicable in well-marked cases of both horizontal and vertical deviations in patients of all ages, provided that the examiner is sufficiently adaptable to children.

Another test for well marked cases is to move the finger or a light from one side of the field of vision to the other and back again, having the patient follow it with his eyes. If one eye lags behind the other as they turn in a certain direction, the muscle that is acting is palsied, and if the eye stops in the middle line the paralysis is nearly or quite total. This test can be used in both children and adults, as the eyes of a baby will follow a small electric light, and is applicable to both horizontal and vertical deviations.

If neither eye will fix the object the deviation is either simulated or hysterical.

Diplopia is present and annoying in paretic deviations when the patient is old enough to appreciate it, has possessed binocular single vision, and the condition has not existed too long, while it is very rare in strabismus. Diplopia in which the distance between the double images increases as the eyes are turned in one direction and decreases as they move in the opposite way is almost certainly diagnostic of muscular paresis, especially when it is accompanied by vertigo and faulty orientation, though its absence is not so reliable an indication. Whenever it is possible for a patient with paresis of an ocular muscle to do away with the diplopia and secure binocular single vision by twisting his head into any position, he will assume it habitually, no matter how awkward or inconvenient such a posture may be, so a habitually distorted position of the head should be enough to suggest a possible paresis of one or more muscles of the eye. The most pronounced cases of this ocular torticollis are met with in paralysis of the superior oblique. Such a pose will not be taken unless binocular single vision can be obtained thereby, or before the development of the faculty.

Other diagnostic points are that paretic deviations usually are constant, seldom of a fleeting nature, and never alternating, while strabismus may be constant, alternating, or intermittent. If we have an opportunity to examine the eyes during sleep we find that a strabismus has disappeared, while a paretic deviation persists. Amblyopia of the deviating eye increases the probability of strabismus, but does not exclude paresis. The coexistence of pareses in muscles that are not ocular, or of other symptoms which point to a lesion of the central nervous system, greatly increases the probability that the deviation is paretic. Most, but not all vertical deviations are due to muscular paresis. A convergent strabismus in a child may decrease as he grows older until the eyes become nearly straight, or pass over into divergence, but binocular single vision never is attained and the condition cannot be affected by medication, while a muscular paresis may disappear spontaneously, or as the result of medical treatment, with the restoration of binocular single vision.

After we have made this differentiation a number of other observations have to be made.

STRABISMUS

We note the direction of the deviation. If the visual line of one eye is inclined toward the nose the strabismus is **convergent;** if it is inclined outward the strabismus is **divergent;** if it inclines up or down we have a case of **vertical** strabismus. A great advantage of the scientific nomenclature of these conditions, which has been urged for adoption, is that the terms describe much more exactly the direction of the deviation. The general term for strabismus is heterotropia. Convergent strabismus is esotropia; divergent strabismus is exotropia; vertical strabismus in which one eye turns up is hypertropia, one in which an eye turns down is hypotropia. Vertical deviations almost always are more or less out or in as well as up or down, and according to *Hansell* nearly every case of esotropia is associated with more or less hypertropia; these variations are indicated by such compound terms as hyperesotropia and hypoexotropia. In spite of this manifest advantage the mental effort to distinguish the meaning of words which resemble one another so closely, both when written and when spoken, as hypertropia, hypermetropia, and hypotropia, may act as a deterrent to its universal adoption. Such a mental effort hampers the close following of a line of thought, and this is the reason it is not adopted here.

A **symblepharon** may draw an eye to one side, when the patient will suffer from diplopia and its attendant symptoms, the same as in muscular paresis, if the sight has not been impaired too badly; this cause is perceived readily. A convergent or divergent deviation may follow an **operation for pterygium** through the contraction of cicatricial adhesions; the history and the presence of a cicatrix guide us to the correct diagnosis.

We know little concerning the ætiology of a vertical strabismus, except that it may be due to too short a superior or an inferior rectus. Such a case is rarely seen. The most common form of strabismus is the convergent, the divergent is met with much less often, and the causes of these deserve separate consideration.

Causes of Convergent Strabismus

Some persons can simulate convergent strabismus for a time, but a careful inspection will reveal that both eyes converge, that neither looks straight forward. The same is true of hysterical convergent

strabismus, which is caused by the spastic contraction of the two interni. When these cases have been excluded, together with those in which a deviation is produced by a cicatricial contraction, and the rare ones in which it follows the loss of sight of an eye, we may say that convergent strabismus appears in children from three to five years of age apparently as the result of several factors, no one of which seems to be sufficient alone, while the part played by each appears to differ in individual cases. Factors upon which stress has been laid are hypermetropia, amblyopia of the squinting eye, anatomical faults in the positions of the orbits, too short an internus, too weak an externus, spasm of the convergence, absence of the power of fusion, and the presence of esophoria either alone or combined with hyperphoria, or with a declination of the vertical meridian of the eye.

Very few if any question the established fact that **hypermetropia** is present in the great majority of cases of convergent strabismus, just as myopia exists in a large proportion of persons whose eyes diverge, and this refractive error has been assumed to be the principal cause, especially when the errors in the two eyes were quite different, or the astigmatism in one rendered its vision decidedly the poorer. It cannot be doubted that this is an important factor, and one that probably is primary at times, for if we correct the errors properly under atropine we may cure the strabismus, though not commonly; in most cases it is lessened in degree, but in some it is not affected. Paralysis of the accommodation without correction often lessens and sometimes obviates the squint for the time, while in other cases it fails to do so, so the importance of this factor must vary. Most people who do not squint are hypermetropic, or have hypermetropic astigmatism, so these errors alone cannot be held to be responsible.

The question whether an **amblyopia** of the strabismic eye is the cause or the result of the deviation is one which has excited much controversy. It certainly is a factor in the maintenance, even though it may not be the cause of a squint, for it has been demonstrated that the visual power of such an eye sometimes can be improved by making it work, and that measures taken with this in view, together with the correction of the refractive errors in both eyes, greatly facilitate the development of binocular single vision and the doing away with the strabismus. At the same time we occasionally meet with a person who has a considerable degree of amblyopia in one

eye, but has never shown any appreciable squint, so this cannot be the sole cause.

Such **anatomical faults** as an abnormal position of the orbits, or too short an internal rectus, are possible causes, and are to be considered primary when they are present, but they are not common. We should expect a strabismus due to an anatomical fault to persist during sleep or unconsciousness, but this is a very exceptional occurrence. When an internal rectus is too short, or an external rectus too weak, the abduction is faulty, but this power is normal in a large proportion of the cases, *Roemer* says in eighty per cent. *Wootton* has shown that an insufficiency of divergence may be the main factor in some cases. He succeeded in obtaining binocular single vision in children thirteen years old who exhibited this fault, and in whom correction of the refraction did not suffice, after advancement of the external recti. It has been claimed that an **esophoria** may change into an esotropia, and this is understandable when the internal rectus is innervated excessively through the association of the convergence with the accommodation of a hypermetropic eye, but it seems as if a change of that nature in such a case must be subordinate either to the hypermetropia, or to the fact that the two eyes do not work together. It is difficult to understand how an esophoria, whether combined or not with a hyperphoria, or with a declination of the vertical meridian of the eye, can change into an esotropia, or convergent strabismus, when the eyes are emmetropic and have the power of binocular single vision. If the esophoria is reinforced by such a powerful factor as hypermetropia, or loss of the power of fusion, and strabismus is developed, the question still remains whether the esophoria is not a relatively unimportant, rather than the primary cause of the strabismus.

The power of **binocular single vision** is that of fusing the impressions communicated to the brain by the images formed on the retinæ of the two eyes so that the object is seen as one. We have reason to believe that this power does not exist at birth, but develops later and becomes complete at some time in early childhood, probably between the second and the fourth years. How it develops we do not know; we may suppose that the faculty lies dormant and awakes about this time, or that certain nerve tracts in the brain are incomplete at birth and develop gradually to make this power possible when they have reached their full development. We know that older children do not necessarily possess this power, that with

patient endeavor we may succeed in getting children in whom this power is apparently absent to observe the two images, one of which they have been accustomed to suppress, and in superimposing them without fusion taking place, and if we are persistent we may possibly succeed in getting these images to blend and in establishing binocular single vision. It appears also that the sooner such an attempt is made after the eye starts to turn, *i.e.,* the younger the child, the more likely we are to obtain such a result. Yet it is not certain that this power is not in abeyance sometimes in convergent strabismus, it seems to vary much in strength, and its absence does not always cause an eye to deviate.

It seems probable that the two main factors in the production of the great majority of cases of convergent strabismus are ***absence of binocular single vision*** and ***hypermetropia,*** either combined or not with astigmatism, and that these always are reinforced by other factors which may be held to play subordinate parts, as a rule, although in isolated cases each one of them may be paramount.

Causes of Divergent Strabismus

The actuating causes of divergent strabismus are not thoroughly understood, but they appear to be mechanical. The condition usually appears in children about the age of ten or twelve. As a rule, we are unable to learn whether binocular single vision existed or not before one eye began to diverge, but occasionally a prior examination has demonstrated its presence, perhaps that it was weak or imperfect, and usually that the visual axes tended to diverge when the eyes were at rest, technically that the patient had exophoria. Even when binocular single vision is absent divergence does not develop until some other factor acts, and it is questionable whether it develops at all when this power is perfectly developed, but when the power is weak certain **mechanical conditions** appear to transform the exophoria into an exotropia. The most common actuating factor is a preponderance of the action of the external rectus over that of the internal. When the external rectus is too short the strabismus appears at an earlier age and the rotation of the eye inward is impeded. The action of the external is favored over that of the internal rectus by a great divergence of the axes of the orbits, and by an elongated eyeball, conditions which are apt to be present in myopia, from which the majority of these patients suffer. We may suppose also that as the demands upon the accommodation and convergence

are less than normal in myopia, the internal rectus loses its strength in comparison with its opponent, which has its usual duties to perform. Sometimes the external rectus is found to be disproportionately thick and strong, and it frequently happens that this muscle seems to increase in relative strength as age advances, so that an eye which converged in a child may be seen to diverge in adult life, and when the sight of an adult's eye is impaired too much to be of use any longer divergent strabismus is apt to appear.

Observations to be Made in Strabismus

After noting the age of the patient, whether the strabismus is constant, intermittent, or alternating, we should measure the degree of the deviation before doing anything else. Then we should determine the vision and refraction of each eye, ascertain the degree of any amblyopia that may be present, learn whether binocular single vision is frankly present, and if not try to elicit a perception of an object by both eyes at the same time, and then see if the images can be made to fuse. The greatest deviations of the eyes are to be seen in convergent strabismus, those of the divergent form are less on the average, while those of the vertical are slight. Ordinarily we perceive strabismus from the relative position of each cornea, iris, or pupil to the median vertical plane of the face, or to the horizontal plane, and we may learn by practice to estimate roughly its degree from these relative positions, or by having the patient fix on one of our fingers held at first in front of our own eye and then moved outward until the squinting eye seems to look straight into ours, but such methods depend wholly on the judgment of the examiner and are not accurate. *Priestley Smith's* **tape measure test** is a modification of the latter in the interest of accuracy, which it approximates, but does not attain perfectly. An exact distance is maintained by means of a tape one meter long, one end of which is held by the patient just beneath his eye, while the other end is attached to a ring which is slipped over a finger of the hand of the examiner that holds an ophthalmoscope with which he reflects light into the patient's deviating eye. Another tape marked off in degrees is attached to the same ring and is carried over a finger of the examiner's other hand upon which the patient fixes while it is moved outward until the reflex from the light thrown by the ophthalmoscope appears on the center of the cornea of the squinting eye.

The number on the tape at which the moving finger stops gives the degree of strabismus.

Another simple, though not absolutely accurate method is to measure the degree with the aid of the **strabismometer.** The good eye is covered, the squinting one made to fix on a distant object, the instrument is placed on its lower lid so that zero is exactly beneath the center of the pupil, the good eye then is uncovered and caused to fix on the same object, when the degree of deviation can be read off the instrument. The same result is obtained if we make a mark on the lower lid below the center of the pupil or one edge of the cornea, in each of these positions and measure the distance between them in millimeters. One great advantage of both the tape measure and the strabismometer tests is that they can be used even with very young children.

With older and intelligent patients a much more accurate measurement can be obtained with a **perimeter** and a small electric light. The patient fixes the central spot of the instrument while the examiner passes the light, together with his own eye, along the arc until the light reflex appears on the center of the cornea of the squinting eye; the perimetric reading gives the angle of strabismus plus or minus the angle γ, which remains to be determined. The good eye is covered and the squinting one fixed on the central spot, close to which the light is brought. If the light reflex is on the center of the cornea when the light and the central spot coincide, there is no angle γ; if it is not we move the light to the right or left until the reflex is central and determine the angle from the degree of the arc on which it rests. If it is to the temporal side of the eye the angle γ is positive and its degree is to be added to that of the strabismus already obtained; if it is to the nasal side it is negative and must be subtracted.

A **prism test** is possible only when binocular vision is present. A red glass is placed before one eye and, when diplopia is maintained, one prism after another is slipped into place with their bases in the direction opposite to that of the squint until one is found with which the images are fused, or exactly superimposed; one half of the degree of this prism is the degree of the angle of strabismus. This test is of particular value in a few cases in which the angle γ is so large that a strabismus is counterfeited. A case in point is that of a man who desired to have an operation performed to correct a slight deviation of his eyes which really constituted somewhat of a

deformity, but this test revealed that he had binocular single vision, orthophoria, and that his angle γ was unusually large.

MUSCULAR PARESIS

A patient with paresis of one or more of the extrinsic muscles of the eye has a secondary deviation that is much greater than the primary, impairment of motility in the direction of the affected muscle or muscles, diplopia with vertigo and faulty orientation if the paresis is recent, and sometimes an abnormal position of the head. The paresis may be due to disease of the muscular tissue itself, to a lesion in the orbit, or to one that compromises the nerve supply anywhere along the course of the nerve, or in the brain. It may vary from extremely slight, when there is no apparent deviation, to total paralysis, and may affect a single muscle, a group, or all of them.

When the paresis is very slight the only symptom may be **diplopia when the eyes are turned far** toward the limit of the binocular field of fixation in the direction in which the affected muscle is called most strongly into action. In such a case we need to learn which muscle is at fault. If the patient sees double only when he looks far to the left we know that either the right internal rectus, or the left external rectus is acting badly, as they are the two muscles which take part in this movement of the eyes. To ascertain which of the two is affected we place a red glass before one eye and have him look at a light placed far to the left, when he will see one red and one white light. If the red glass is in front of his right eye and the red light is to the left of the other the diplopia is crossed, the eyes diverge, and therefore the right internal rectus is paretic, but if the red is to the right of the white light the diplopia is homonymous, the eyes converge, and therefore the left external rectus is too weak to perform its duty completely. We must be on our guard against a hysterical diplopia of this nature that is met with occasionally. A lady exhibited diplopia at the extreme right of the field, and had been operated on several times for heterophoria. She was known to be suffering from hysteria, and finally this was demonstrated to be the origin of her muscular troubles by obtaining diplopia at the extreme right, then slowly carrying the light upward and then across the upper limit of the field to the extreme left while the diplopia was maintained throughout without change, a phe-

nomenon that could be accounted for by no possible combination of muscular defects.

When a patient complains of **diplopia, vertigo,** and an **inability to determine which of the two images he sees is the true one,** or **carries his head twisted to one side** to do away with these annoyances, we have to note whether one eye deviates in any direction when his head is in its normal position, whether the diplopia is crossed or homonymous, in which eye and in what direction the secondary deviation appears, and the position of the head when this is abnormal. If we know the actions of the individual muscles, singly and combined, we can deduce from these symptoms which one or ones are affected, but it is of little use to try to memorize the almost numberless combinations that may take place.

If the patient holds his head ***erect, but turned to one side,*** and has diplopia when he looks straight forward with his head in its normal position, either the external rectus of the eye on the side toward which the head is turned, or the internal rectus of the other, must be paretic, because these two muscles are relieved greatly from duty in that position of the head, and we know the condition to be paresis rather than paralysis because the function of the muscle is not abolished. To determine which muscle is at fault we use the same test as when the paresis is so slight that diplopia appears only when the eyes are turned to the extreme limit of the field, except that we place the light directly in front of him. If the red glass shows that the diplopia is homonymous the internal rectus is paretic, if the diplopia is crossed the external rectus is too weak. We confirm the results of this test by covering the eyes alternately and observing the deviation and the movement of adjustment to fix the light as these appear in each; the sound eye has the greater or secondary deviation and makes the longer excursion in order to fix, as has been explained. Thus we learn which eye, and whether its external or its internal rectus, is affected, even when the paresis is so slight that the deviation can scarcely be seen and the patient is able to secure binocular single fixation by a simple rotation of the head.

When the paresis is more marked, and when paralysis is present, the eye deviates away plainly from the affected muscle, its movement toward it is restricted when it tries to follow a moving object, the secondary deviation of the sound eye is enormous, and the diagnosis is easy.

When a patient inclines his head on one shoulder, lowers his chin and rotates it toward the same side, taking the position of ***torticollis,*** we should think of a possible paralysis or paresis of either the superior oblique of the eye on the opposite side from that to which the head inclines, or the inferior rectus of the other. Such a condition certainly is present if the patient has vertical diplopia when the head is held erect. One of the two images is tilted and the vertical distance between them increases as he looks down. The diplopia in paralysis of the superior oblique is slightly homonymous, that in paralysis of the inferior rectus is slightly crossed, and in both the upper end of the lower image tilts toward the upper, so the pictures produced by these two conditions resemble each other closely. The differentiation is made through observation of the secondary deviation, which takes place in the sound eye and leaves the diagnosis made by exclusion. Whether the paresis is sufficient or not to cause the patient to assume the extreme degree of torticollis, we have to consider the exact direction of the secondary deviation, which is downward and slightly outward in paresis of the inferior rectus, downward and slightly inward in paresis of the superior oblique, and the limitations of movement, which are most marked downward and inward in the former, downward and outward in the latter.

When a patient goes about with his ***chin raised and his head tilted*** a little to one side in order to avoid a vertical diplopia in which the upper image is tilted, its upper end leaning away from the other, he has a paralysis or paresis of either a superior rectus or an inferior oblique. Crossed diplopia points to the rectus, homonymous to the oblique, but it is crossed sometimes in paresis of the latter, perhaps because a latent exophoria has been made manifest. We exclude one eye by observation of the secondary deviation, which is always in the good one, and differentiate between the two muscles by noticing the excursions of the other. If it lags behind when the patient looks up, up and in, and especially up and out, the superior rectus is at fault, while if the eye rises freely up and out, but lags badly when it tries to move up and in, the inferior oblique is paretic.

The following table, copied from *Duane,* is of great assistance in locating the affected muscle in cases of slight paresis as soon as we have become sufficiently familiar with the abbreviations, so that their interpretation does not entail too great a mental effort. The abbreviations mean:—

E, eyes;
R, turned to the right;
L, turned to the left;
u, turned up;
d, turned down;
DH, homonymous diplopia;
DX, crossed diplopia;
DR, Vertical diplopia with the image belonging to the right eye below;
DL, Vertical diplopia with the image belonging to the left eye below;
> greatly, increasing progressively and fast.

Thus the abbreviation Eu & r DL > greatly means a vertical diplopia with the image belonging to the left eye below which increases progressively and fast when the eyes are turned up and to the right, literally eyes up and right, diplopia left increasing.

DIPLOPIA	PARESIS OF
Er. DH > greatly	right external rectus
Er. DX > greatly	left internal rectus
El. DH > greatly	left external rectus
El. DX > greatly	right internal rectus
Eu & r. DL > greatly	right superior rectus
Eu & r. DR > greatly	left inferior oblique
Eu & l. DR > greatly	left superior rectus
Eu & l. DL > greatly	right inferior oblique
Ed & r. DR > greatly	right inferior rectus
Ed & r. DL > greatly	left superior oblique
Ed & l. DR > greatly	right superior oblique
Ed & l. DL > greatly	left inferior rectus

When all of the muscles supplied by the third nerve are paralyzed we first notice ptosis. After we lift the lid we see that the eye is drawn outward and a little downward by the unaffected externus and superior oblique, that there is a slight degree of exophthalmos, that the pupil is dilated, and that it does not react to light. The power of accommodation is lost and the diagnosis is made quickly and easily.

When the same picture is presented except that the eye looks directly forward and is immovable, the case is one of total ophthalmoplegia. If the iris and ciliary muscle continue to act the condi-

tion is one of external ophthalmoplegia. Internal ophthalmoplegia is the condition when the iris and ciliary muscle are paralyzed while the extrinsic muscles are not.

The cases of ocular palsy that we meet with in practice are not always analyzed so easily, because several and varying muscles may be affected at the same time, conditions may change frequently when the pareses are of a fleeting nature, and atypical pictures may be produced after a paralysis has been long in existence. Secondary contractures may form in the antagonists to the paretic muscles, the conditions of which may grow better or worse, or the paretic eye may fix while the sound one takes the position given it by the secondary deviation. We also meet with conditions in which only certain functions of associated muscles are abrogated, as in conjugate paralysis, paralysis of convergence, and paralysis of divergence. In the first both eyes are unable to move in a certain direction, in the second they cannot converge.

The commonest form of **conjugate paralysis** is the lateral, in which the eyes turn freely to the right, or to the left, but are arrested at the median line when they try to turn in the opposite direction, as though the external rectus of one and the internal rectus of the other were paralyzed. Such a condition is possible, so it has to be excluded by testing the convergence, when the apparently paralytic internal rectus contracts freely in association with the internal rectus of the other eye, proving that the muscle itself is not paralyzed. Both eyes are apt to be deviated and the patient to carry his head rotated in the same direction.

Conjugate paralyses upward or downward are less common. They may or may not be associated with diplopia, and the patients carry their heads tilted either backward or forward. Occasionally the eyes will move neither upward nor downward.

In **paralysis of convergence** the eyes rotate freely in all directions, but cannot converge to fix on a near object. The eyes remain straight as an object is brought close to them, but there is no diplopia because the images of the object fall upon corresponding points in the two retinæ.

Sometimes a convergent deviation appears suddenly together with a homonymous diplopia in which the images are farthest apart when the patient is looking straight forward at an object in the distance, but approximate each other as the eyes are turned to either the right or the left, and especially as the object is brought nearer.

This is **paralysis of divergence.** In such a case the images fuse when the object is brought to within a few inches of the eyes. Paresis of the external recti is excluded by the fact that the eyes rotate freely to the right and the left. When the deviation of the eyes diminishes as the object of fixation is brought nearer we are able to exclude spasm of the convergence, as we can also by the stable character of the condition, but after a paralysis of divergence has lasted for some time a secondary convergence excess may follow, and may take the form either of a spasm, or of a continuous overaction that causes the deviation and the diplopia to be more nearly alike at all distances. Paralysis of divergence may be associated with spasm, paralysis, or conjugate paralysis of the vertical muscles, or with paralysis of convergence. *Duane* claims that these cases are not so very rare.

Finally we may meet with a rare congenital anomaly, or rather combination of anomalies, that cause one or both eyes to deviate peculiarly. The symptoms which are associated to form a characteristic syndrome are tabulated in the following manner by *Duane:*

1. Complete, less often partial, absence of outward movement of the affected eye.
2. Partial, rarely complete deficiency of movement inward.
3. Retraction of the eye into the orbit when adducted.
4. A sharply oblique movement of the eye either up and in, or down and in when adducted.
5. Partial closure of the lids when the eye is adducted.
6. Paresis, or at least deficiency of convergence, the affected eye remaining fixed in the primary position while the sound one converges.

When the patient holds his head erect and looks straight forward his eyes may not deviate, may converge, may diverge, or he may have diplopia when he turns his eyes in a certain direction. Secondary deviation may equal or exceed the primary, but one case is reported in which it was wholly absent. The patient complains of asthenopia, headache, a sense of strain, and often of diplopia, which almost always can be elicited by tests when it is not spontaneous, and frequently is avoided by a rotation of the head in one direction or another, yet he rarely suffers from vertigo or false projection. The vision varies from normal to very poor, but the accommodation and the reactions of the pupils remain normal. A very striking symptom is the retraction of the eye back into the orbit

whenever the eye turns in, but this is not always present, and sometimes is replaced by a protrusion of the globe. The closure of the lids is not a ptosis dependent on the recession of the eyeball, for the lower lid rises as the upper one descends; it is not always present, and sometimes the palpebral fissure widens instead. The degrees of all of these symptoms vary, and it is not necessary that all of them should be present in any individual case. The external rectus has been demonstrated to be absent or replaced by a cord of connective tissue in several cases, but we cannot stop to discuss the various theories which have been advanced to account for the various symptoms.

Differentiation of Lesions Productive of Paresis of the Ocular Muscles

When there is a constant great deviation of the eye of a baby, or when such a congenital condition has proved permanent in after life, no actuating lesion can be found, and no cerebrospinal symptoms develop, it is possible that the muscle which should turn the eye into position is congenitally absent, or that there is an aplasia of the nucleus of the supplying nerve. Such a condition is an extremely rare anomaly.

A paresis due to disease of the muscular tissue itself likewise is very rare. Its diagnosis must depend on the presence of a similar disease of muscles elsewhere with the exclusion of any lesion of its nerve supply, and at best can hardly be more than probable. If a patient presents a recent deviation of both eyes that cannot be ascribed to any lesion of the nerve supply, has a doughy œdema of the lower lids, and exhibits other symptoms of trichinosis, we may be justified in the conclusion that trichinæ have invaded and destroyed more or less of the tissue of these muscles.

Muscular paresis is more likely to be caused by trouble in the orbit, or by disease of the nerve. The presence of a wound points to a direct injury of either the muscle or the nerve, while exophthalmos, local pain, tenderness or inflammation lead us to seek the cause of the paresis in the orbit or in the accessory sinuses. Paresis of the superior oblique will follow an operation for frontal sinusitis when the situation of the trochlea has been disturbed.

A paresis of one or more of the muscles of the eye when associated with symptoms of cerebral or cerebrospinal trouble, furnishes

aid that is more or less valuable in different cases in locating the exact site of the lesion in the central nervous system, and sometimes the muscles affected give us a hint as to its nature. We speak of a lesion that compromises the nuclei as nuclear, of one that affects the nerve anywhere else along its course as peripheral, of one that impairs the higher centers as supranuclear. A peripheral lesion along the base of the skull is called basal. We cannot always tell whether a lesion is peripheral or nuclear, but the following points may be of assistance.

Peripheral Pareses

When the patient presents a paresis of all of the muscles supplied by the third nerve, associated with either no cerebrospinal symptoms, or with symptoms indicative of trouble at the base of the brain, we should inquire very carefully in regard to **acquired syphilis,** for this is its most common cause. It rarely happens that hereditary syphilis acts in this way, and the external rectus and the superior oblique are much less apt to be affected. But we must remember that while a paresis of all of the muscles supplied by the motoroculi is suggestive of active syphilis, a paresis of one or more individual muscles supplied by it is more likely to be caused by tabes dorsalis.

The next most common cause of a peripheral palsy is **rheumatism,** when we often can elicit a history of exposure to cold and damp. The external rectus is the muscle most apt to be affected, and often this affection is associated with a facial paresis. Whether the muscular tissue or the nerve is affected we cannot tell with certainty, but we have reason to believe it is the nerve. Such rheumatic palsies have been followed years later by tabes, disseminated sclerosis, and paralytic dementia, but they can scarcely be looked upon as early symptoms of such diseases.

The abducens is particularly apt to be implicated in a fracture of the base of the skull. A rapid succession of troubles in the motor nerves of the eye, as well as in the optic, facial, trigeminal and olfactory, is an almost certain indication of a rapidly progressing **lesion at the base of the brain.** An ocular paralysis with homonymous hemianopsia, or an optic neuritis, is not diagnostic, but is suggestive of a basal lesion, while one with temporal hemianopsia, anæsthesia of the cornea, a neuroparalytic keratitis, or a trigeminal neuralgia, is almost certainly of basal origin. A basal lesion is

excluded when the third nerve is affected with the exclusion of the branches that supply the iris and ciliary muscle, so that the activity of these muscles alone remains intact.

Paralysis of the abducens and facial on one side and of the limbs on the other side of the body locates the lesion as lying in or close to the **pons.** A third nerve paralysis on one side with hemiplegia on the other indicates that the lesion is either in the lower part of the **peduncle,** or in its near vicinity at the base of the brain; the former is the site if the iris and ciliary muscle are not affected.

Most of the palsies of the ocular muscles met with in cases of acute infectious disease and of poisoning are nuclear, but it is probable that some of them are peripheral.

Nuclear Pareses

If one muscle after another supplied by the motoroculi becomes paretic in an order that corresponds to the anatomical arrangement of the nuclei, there is no doubt that the lesion is nuclear. Isolated pareses of several muscles supplied by this nerve that cannot be explained by trouble in the orbit and are not accompanied by symptoms of tabes, indicate small lesions in the nuclei, which often are caused by arteriosclerosis, though the paresis of a single branch may be due to a basal affection. Paresis of all of the external muscles supplied by the motoroculi, whether the external rectus and the superior oblique are involved or not, while the muscles within the eye remain intact, indicates nuclear trouble with a high degree of probability, especially when the condition is bilateral, as does also paresis of the internal muscles with exemption of the external, but when all of the branches of the motoroculi are involved, and in total ophthalmoplegia, the lesion may be in the nuclei or elsewhere.

A nuclear palsy may be **congenital** from aplasia of one or more of the nuclei. In such a case the patient has no diplopia, the secondary deviation is not pronounced, the paresis is stationary, and the condition, which is not symptomatic of disease of the central nervous system, is apt to be hereditary, and to be accompanied by other congenital anomalies either in the eye, or in other parts of the body. Total paralysis from this cause cannot be distinguished from that due to congenital absence of the muscle in any way except by cutting down upon and examining the latter.

Acquired nuclear pareses may be acute or chronic. When the

muscles of an eye become paralyzed rapidly we have to determine first whether the trouble is the result of traumatism, intoxication, or infection. The onset often is accompanied by fever and convulsions, while the history is likely to be of great assistance when it can be obtained. In the absence of a history we look for a bruise on the head, if we find one we investigate for a fracture of the base or of the orbit, and look for basal pareses of other nerves; if neither of these can be found we conclude the trouble to be nuclear. A hemorrhage or softening in the region of the nuclei may follow a blow on the head without fracture. Such cases are comparatively rare.

Intoxication is a more common cause when both eyes are affected. Alcohol may induce an acute hemorrhagic polioencephalitis, in which we find the extrinsic muscles of the eyes pretty generally paretic, the patient dizzy, staggering, and suffering from headache, delirious, or in a more or less comatose state. In these cases ptosis often is absent, and the internal muscles of the eyes are not affected, as a rule. The smell of the breath is not to be relied on in making the diagnosis, because liquor often is given as a remedy in all manner of sickness before a physician is called, but the general physiognomy characteristic of many hard drinkers may be of assistance. Sometimes we find an optic neuritis with hemorrhages in the retina, and an abnormal paleness of the temporal side of the papilla frequently is due to the habitual abuse of alcohol and tobacco.

Ptomaine poisoning may produce a similar condition, but in this the internal muscles are always paralyzed, and there is ptosis. The other muscles may or may not be affected, according to the severity and duration of the attack, but the bilateral ptosis with internal ophthalmoplegia is the important feature when taken in connection with the general symptoms of the disease.

In acute nuclear paresis due to poisoning with carbonic acid, lead, and snake bite the grave cerebral symptoms of alcoholism are absent, though headache and convulsions may occur, the internal muscles are apt to be involved while some of the external ones escape, and an optic neuritis more often is present.

When traumatism and intoxication can be excluded an acute ophthalmoplegia must be due to ***infectious*** or ***central disease.*** It may be associated with acute poliomyelitis, or with bulbar paralysis. Tuberculosis or syphilis may excite an acute hemorrhagic

polioencephalitis which produces an ophthalmic picture like that of alcoholism, but they do so with great rarity. An acute paresis of the external rectus often is associated with facial paresis, one of the other muscles only occasionally. When the accommodation is paretic although the pupil reacts properly to light, we can say almost with certainty that the patient has suffered recently from diphtheria, but a similar affection of the extrinsic muscles rarely is caused by this disease; in these rare cases the external rectus is the muscle usually affected. The pareses that follow influenza are extremely variable because they are not necessarily nuclear. It is said that the accommodation alone of both eyes may be paralyzed, as is common after diphtheria, but more commonly the reaction of the pupil to light also is lost, with or without a paresis of the extrinsic muscles. Cases have been reported in which only one eye was affected, and in these the lesion cannot have been nuclear. Acute ophthalmoplegia has been known to follow other acute infectious diseases, such as typhoid fever and chicken pox, as well as to occur in septicæmia and diabetes. Usually we are able to determine the cause of an acute ophthalmoplegia, but we meet with cases in which we cannot. Sometimes the condition exists alone, or in combination with symptoms that do not aid us in ascertaining either the site, or the ætiology of the trouble, which may be either permanent or transient.

A chronic, progressive nuclear ophthalmoplegia may appear entirely by itself, beginning while the patient is young with a slight ptosis and a little impairment of the functions of the extrinsic muscles, both of which increase slowly as he grows older, perhaps with remissions and regular recurrences. Both eyes are affected, though the trouble may begin in one before the other and the ophthalmic branch of the facial nerve frequently is involved. No morbid changes are to be seen in the fundus, and the cause is unknown. This condition is rare, and the existence of any disease of the central nervous system must be carefully excluded before the diagnosis of its presence can be made in any case.

A chronically progressive ophthalmoplegia with troubles of mastication and swallowing indicates bulbar paralysis. Sometimes the addition of a hemiplegia aids in locating the lesion more exactly.

Slowly developing pareses of the ocular muscles are indicative of **tabes** in most cases, and when they are accompanied by a reflex immobility of the pupils and a progressive optic atrophy, the diag-

nosis is almost as good as made, though such a combination may be seen in general paresis complicated by tabes. An important point is that the onset of the muscular paresis may antedate that of the other symptoms by a long interval, even one of several years. Syphilis is the underlying cause in a very large proportion of the cases of chronic progressive nuclear palsy of the ocular muscles, while tuberculosis is the fundamental disease in many of the rest.

Occasionally we meet with such a nuclear palsy in syringomyelia, progressive muscular paralysis, the combined diseases of the posterior and lateral columns of the spinal cord, paralysis agitans, disseminated and multiple sclerosis, diabetes, and exophthalmic goiter. The diagnosis is made in all of these from the combination of the palsies with the symptoms of the causal disease.

Conjugate Paresis

A conjugate paresis may be produced by a **supranuclear lesion** in the great ganglia of the brain, the crura, pons, corpora quadrigemina, or optic thalami, or by a **lesion** situated **in the cortex** of the opposite side of the brain, and may be either irritative or destructive. If the limbs on one side of the patient's body have convulsive movements it is irritative; if they are paralyzed it is destructive. If the lesion is irritative and the eyes deviate toward the side on which the convulsions occur, it is situated in the cortex, but if they turn in the opposite direction it is supranuclear. Conversely, when hemiplegia is present both eyes deviate toward the paralyzed side if the lesion is supranuclear, and away from it when the lesion is cortical. In paralysis the head is rotated in the same direction as the eyes.

Many supranuclear pareses are evanescent in character, but when the lesion is situated in the pons, or involves the nucleus of the sixth nerve, it usually is permanent. Generally we find a facial paresis present on the same side with the affected external rectus. Conjugate palsies of the horizontally acting muscles are not infrequent in gross lesions of the cerebrum and pons. Those of the vertically acting muscles may be caused by lesions in the cortex, or by disease of the corpus striatum or optic thalamus, but our knowledge of the sites of the lesions which induce these conjugate palsies is almost if not quite purely theoretical. *Hansell* and *Reber* report a case in which a conjugate palsy of this nature preceded for some years a progressive ophthalmoplegia and optic atrophy with incoordination

of the limbs and exaggerated knee jerks. Further points that may lead to the determination of the site of the lesion in a case of conjugate palsy may be obtained from the other neurological symptoms present, but for a consideration of these the reader is referred to the textbooks on neurology. We have to remember that any form of this deviation may be of hysterical origin.

Paralysis of Convergence

Paralysis of convergence is due to a lesion that involves the convergence center, which is supposed to be located in the fissure of Sylvius. It may be associated with paralysis of the accommodation, or of the vertically acting muscles, but occasionally it occurs alone, and then possibly it may be caused by cerebellar disease. It is met with most commonly in disseminated sclerosis, but at best is quite a rare condition.

Paralysis of Divergence

The cause of paralysis of divergence is not known. Some of the cases reported were associated with brain lesions, while the suddenness of development and the permanence of the symptoms seem to indicate in others localized hemorrhages, as suggested by *Duane*. In a few cases it seems to have started as a paresis of the externus, in a few others to have passed into such a muscular paresis, so it would seem probable that all are due to lesions in the brain, but their sites cannot as yet be determined.

CHAPTER VI

Motor Anomalies without Deviation of the Eyeballs

An imbalance of the ocular muscles that does not induce a visible deviation of the eyeballs is known as **heterophoria,** which is divided into several varieties. A disposition on the part of the vertically acting muscles to raise the visual axis of one eye above the horizontal plane is **hyperphoria,** a tendency to direct it below this plane is **hypophoria,** or better **cataphoria.** When the disposition on the part of the laterally acting muscles is to make the visual axes cross too soon we speak of **esophoria,** and when the axes tend to intersect too far away we call the condition **exophoria.** In a great many cases the disposition is toward both a lateral and a vertical deviation, which we designate by terms which indicate the combination formed, like **hyperesophoria,** and **hyperexophoria.** A disposition on the part of either of the oblique muscles to cause the vertical axis of one eye to incline toward or away from that of the other is called **cyclophoria.** All of these errors may be caused by defects in the muscles themselves, or in their innervations, and, with the exception of cyclophoria, they may be caused also by anatomical faults in the construction of the orbits. A perfect balance of the muscles is known as **orthophoria.** Perfect orthophoria is not common, in my experience, and this seems to accord reasonably with the well known fact that the two sides of the face are seldom if ever symmetrically perfect.

Another motor anomaly in which the eyes usually maintain their relative positions to each other is **nystagmus,** in which the eyeballs oscillate together. Careful inspection will reveal that the relative positions are not maintained in quite a number of these cases, that the jerky twitchings of the two eyes are more or less independent of each other, but even when the movements are most independent we should scarcely be justified in considering them deviations of the eyeballs.

HETEROPHORIA

In an uncertain proportion of people any form of heterophoria, in almost any degree, will excite the symptoms of eyestrain, while in a great many, perhaps in the large majority, it produces no annoyance and is discovered only in our routine examination of the eyes. The tests that are employed for its detection may be grouped in three classes: 1, Those in which one or both images seen by the two eyes are displaced; 2, those in which one image is distorted; 3, those in which the image is neither displaced nor distorted. The **prism test,** which is an example of the first class, may be made roughly by holding the prism in the fingers, but much more accurately and conveniently by means of the phorometer, which was devised by *Stevens.* This instrument consists of a base and upright rod to which is attached at right angles an arm furnished with a screw and spirit level by means of which it can be kept perfectly horizontal. Upon this arm is placed a frame containing a pair of 5° prisms which can be rotated by means of a lever. The patient looks with each eye through one of the prisms at a small light twenty feet, or six meters, distant. If we place one prism with its base up, and the other with its base down, the patient will see two images of the light, one directly above the other, if he has binocular single vision, and his eyes have no disposition to swing either in or out; if the upper image is to the right or left of the lower we believe a condition of exophoria or of esophoria to be present, and measure it by the strength of the prism needed to bring the upper image directly over the lower. With the phorometer we rotate the prisms until the images are in a vertical line and read the degree of exophoria or of esophoria from the attached scale. The amount of right or left hyperphoria is learned in the same way when the prisms are placed with their bases in. Modifications of this instrument have been devised, but *Savage* claims that the principle on which they are constructed is faulty as long as the image in one eye is not left undisturbed, and therefore prefers a monocular phorometer.

The second class is represented by the **Maddox rod test,** in which a red glass rod, or a series of red glass rods, mounted in a cell is placed before one eye. This rod has the effect of drawing out the light into a line at right angles to its long axis, so if it is placed horizontally the eye will see a vertical red line, and if this passes

through the light seen by the other eye the eyes balance laterally. In the majority of people the line will be seen to lie to the right or the left side of the light, and seem to indicate esophoria if the line is on the same side with the rod, or exophoria if it is on the opposite side. If the rod is then placed vertically before the eye a red horizontal line appears, which may pass through the light and indicate balance, or be either above or below it and indicate either hyperphoria or cataphoria. The degree of heterophoria can be measured by prisms held before the naked eye, or by means of *Maddox's* tangent scale, which is divided off into degrees and in the center of which the light used in the examination is placed.

The **cover test,** in which a screen is placed before one eye, allowed to remain a moment or two, and then shifted in front of the other, is an example of the third class. This test is of advantage in two ways, it is of both subjective and objective value. If the patient has orthophoria he will see no apparent movement of the object of fixation when the screen is moved, because both of his visual axes have remained directed at the same point when one eye was covered, but if he has heterophoria the object will seem to move, because the patient's eye has moved to regain the position of fixation. After a few trials an intelligent patient can describe the direction and extent of this apparent movement accurately. If it seems to be in the same horizontal direction as that of the screen he has exophoria, if in the opposite direction he has esophoria; if it seems to move downward when the right eye is uncovered, right hyperphoria or left cataphoria is present; if it seems to move upward the case is one of right cataphoria or of left hyperphoria. Frequently the apparent movement is oblique, showing the presence of perhaps hyperexophoria. The apparent movement may be controlled by prisms, and the degree of prism that stops it measures the heterophoria. During this time we observe the actual movements of the eyes themselves. As the screen is carried from one eye to the other no movement will be seen in orthophoria, because the visual axes are parallel, but a movement will be noticed in heterophoria. If the eye moves slightly in the same direction as the screen, exophoria is present; if it moves in the opposite direction, there is esophoria; if it moves upward or downward, the patient has cataphoria or hyperphoria.

The prism and cover tests can be used for the detection of **heterophoria for near,** which is not uncommon in elderly people even when they show little or none for distance, by the substitution of a

black dot on a white surface at the reading distance as the test object. It is absolutely essential in all of these tests that the refractive errors should have been corrected previously, and that the correcting lenses should be worn during the examination.

All of these tests seem to be simple, all have strong advocates, but none of them can be said to be perfectly trustworthy, and the diagnosis of heterophoria is beset by many difficulties. One of these is that after many examinations we may get the eyes to simulate a fault that does not exist. Some years ago I had a patient who had been subjected to the prism test many times by a large number of physicians, and could develop at will 15° of exophoria, or 15° of esophoria; the cover test was new to her and revealed no movement of the eyes and no subjective movement of the object fixed upon. Less marked cases in which the results of a test are apt to be misleading are not very uncommon, and we must not rely on any one method. The only way in which we can be reasonably sure of the diagnosis is to employ every test with which we are acquainted, to compare the findings one with another, and not to decide unless they all indicate some particular form of heterophoria.

A test that is useful as a check in some cases is the determination of the ability of the muscles to overcome prisms with their bases out, in, up, and down. This is to ascertain the **prism convergence,** the **prism divergence,** and the **sursumvergence** of the eyes, which are commonly called the **adduction,** the **abduction,** and the **sursumduction.** The patient fixes his eyes on a small light twenty feet, or six meters, away; a prism is held with its base out in front of one of his eyes until the double images have fused, when it is replaced by a stronger one, and the exchange is continued until the prism is so strong that the images cannot be fused. The strongest prism overcome by the muscle is the measure of the adduction. The abduction is learned in the same way with the prisms held with their bases in, the sursumduction with the bases first up and then down. The normal adduction is from 30° upward, abduction from 6° to 8°, while sursumduction seldom exceeds 2°, but should be equal both up and down. The adduction can be increased to 60°, and the sursumduction to 10° by practice, so such an excess over the normal does not cause us to worry as long as the abduction is normal and the sursumduction is the same in both vertical directions. When the phorometer, or other test, shows a high degree of heterophoria this test should reveal a corresponding

variation from the normal. When the ability to overcome prisms is weak in all directions we are led to suspect a general muscular or nervous weakness due to some cause outside of the eye.

It is well to supplement the information we have now obtained by a study of the field of fixation with the **tropometer,** by means of which the greatest possible voluntary excursion of each eye is determined while the head is immovable. The approximate rotations of the normal eye are given as upward 33°, downward 50°, inward 48° to 53°, outward 48° to 53°. The rotations in all other directions are accomplished by the combined actions of the same muscles that make these four, and can be neglected, as a rule. The line which connects these points after they have been found maps out the monocular field of fixation of the individual. This field varies in different persons, according to their attention and effort, so moderate variations from these figures do not necessarily indicate any abnormality, especially when they are proportionately the same in all directions, but a marked variation in any one direction should be carefully noted. When a fault thus found coincides with one shown by the tests already described there is no doubt as to the diagnosis of one of the ordinary forms of heterophoria, namely exophoria, esophoria, hyperphoria, or cataphoria.

Cyclophoria

If we have the patient look at a straight horizontal line and place before one of his eyes a prism with its base down of such strength that it cannot be overcome, usually one of 5° or 6°, the line will appear to be doubled, and the two lines should be parallel; if they are not he has cyclophoria, but this test gives us no idea of its degree. If we place a Maddox double prism before one eye while the other is left bare the patient will see three lines which should be parallel, but if he has cyclophoria the middle line will not be parallel to the others. If the ends of the middle line coincide with those of the upper and lower ones there is no exophoria or esophoria, and if the mid point of the middle line is equidistant from those of the others hyperphoria is excluded, provided that the double prism is accurately adjusted. Then by substituting a dot for the line the error can be measured by revolving the double prism until the two dots seen through it are in a vertical line. A more convenient method of detecting and of measuring this error is

with Stevens's clinoscope, which consists of two hollow tubes, each with a minute pinhole opening at one end through which the patient can look, and at the other a translucent disk on which is drawn a straight line that runs up from the center in the case of one tube and down in that of the other. The tubes are adjusted on a standard so that they can be placed and kept in the same horizontal plane, as indicated by a spirit level, while they can be moved horizontally, or up and down, as well as made to converge or diverge. The pinhole openings are adjustable to the pupillary distance of the patient, and clips are provided for lenses to correct the refraction. The patient sees the two lines simultaneously, and these should appear to form one straight line. If the line appears to be bent the tubes are revolved about their long axes until it appears to be straight, and then the degree of plus or minus cyclophoria is read from the attached scale. **Plus** cyclophoria means that the vertical axes of the eyes incline from the median plane of the head, **minus** cyclophoria that they incline toward the same plane. Other names that have been used to designate these conditions are plus and minus torsion, and plus and minus declination. When the cyclophoria is either plus or minus in both eyes it is said to be symmetrical; when it is plus in one eye and minus in the other it is nonsymmetrical. In rare cases we may find cyclophoria in one eye, but not in the other.

In the majority of cases we ascribe cyclophoria to a fault in one of the oblique muscles, but *Savage* has pointed out that it may be due to a defect in the innervation centers, and under certain circumstances to faults in other muscles. The symptoms excited by it are those which are described under eyestrain; *Savage* claims that vertigo and nausea are more commonly associated with it than they are with any other form of heterophoria.

Nystagmus

When we see the eyes of a patient quiver as the result of short, rapid, jerky, involuntary, rhythmic movements, we say that he has nystagmus. These movements usually are from side to side, but sometimes they are vertical, rotary, or diagonal. In the great majority of cases both eyes are affected alike, but a few have been reported in which one eye alone was nystagmic. The movements are constant as a rule, though sometimes they are intermittent, but in either case they are apt to increase in rapidity, and perhaps in

range, when the persons attempt to fix, or are excited. Our first inquiry is whether this condition has been present since early childhood, or has appeared at a more recent date, for according to the answer we class it as congenital or acquired. *Lawson* has reported two cases in which a bilateral, horizontal nystagmus could be produced voluntarily, and *Pyle* has seen one in which a unilateral nystagmus was induced in the same way, but the possessors of a power to do this are extremely rare.

We meet with **congenital** nystagmus in cases of albinism, and in other cases in which the vision has been defective since early life. Frequently it is associated with a defective development of the eyes, as shown by the presence of coloboma, cataract, or other developmental faults, and then we may question whether it may not be due to the lack of a proper fovea centralis, so that no distinct point of fixation appears in the field, or to a defect in the coordinating centers that govern fixation. Opacities in the refractive media that have resulted from disease in early life may account for the nystagmus, especially when they are so situated as to lie in the direct line of vision, when the explanation of this effect possibly may be a want of education of the coordinating centers, as similar opacities acquired later in life do not produce the same result. *Lawson* has reported a family in which a hereditary predisposition to nystagmus was transmitted through the females to the males, and states that a few similar cases are on record, so this possibility should be borne in mind when we meet with a nystagmus for which we can discover no cause, but such a case is not of frequent occurrence. Persons with congenital nystagmus seldom if ever complain of any subjective sensations of movement on the part of the objects at which they happen to be looking, a fact that is of considerable diagnostic importance when we are in doubt whether the nystagmus in a certain case is congenital or acquired. *Beard* has suggested that the nystagmus may be a compensatory phenomenon to improve the vision, as if a relatively greater number of percipient elements were brought into requisition at the center of the retina by the rapid oscillations, to compensate for the lack or sparseness in the number or development of these elements.

Acquired nystagmus, in which the rapid apparent movement of objects is a source of much annoyance to the patients, is either a symptom of an organic disease, or the direct result of the person's occupation. When it is due to disease we should make a distinction

between true nystagmus, in which the eyes oscillate together, and **nystagmic twitchings,** in which the movements of the two eyes are not so symmetrical, for *Roemer* tells us that true nystagmus rarely is met with in any disease of the central nervous system except multiple sclerosis, in which he quotes *Uhthoff* as stating that it is present in twelve per cent. of the cases, while nystagmic twitchings occur in a great many diseases, including affections of the labyrinth of the ear, meningitis, hysteria, tabes, alcoholism, paralysis agitans, and diseases of the cerebral cortex. Other authors say that it is fairly common in tumors and other diseases of the cerebellum, as well as in Friedreich's hereditary ataxia, and *Lawson* says that it is one of the symptoms which, taken together with the absence of an optic neuritis, helps to differentiate a posterior basal, from a tubercular meningitis. The same writer says that in the transient disease of infancy called spasmus nutans the shaking or nodding of the head is almost invariably associated with nystagmus in which the movements of the eyes frequently are first toward, and then away from each other. According to *Beard* it is frequent in syringomyelia and sometimes occurs in diffuse myelitis. *Beard* also refers to a case reported by *Malone* in which a vertical nystagmus appeared in a pregnant woman. The full differential values of nystagmus and of nystagmic twitchings, when they appear in various diseases, do not seem to have been worked out completely.

Sometimes a nystagmus can be excited by syringing the external auditory canal with hot water, or by inflating the middle ear with air, in either case with or without inducing dizziness or a faint. This symptom is supposed to depend on an effect produced in the labyrinth, and is utilized by otologists for diagnostic purposes in **labyrinthine troubles.** The direction of the nystagmus is thought to be a guide to the particular semicircular canal that is implicated. In some cases of inflammation of the middle or internal ear we meet with a nystagmus which we are at a loss to interpret, but it is absolutely distinct from that which appears occasionally after the inflammation has extended to the brain.

If we hold our eyes turned as far as possible in a direction in which some of the muscles are taxed to near their physiological limit, twitchings that resemble nystagmus are apt to appear before long, at least in some people, and it has been learned that when such a position of the eyes is demanded habitually in any occupation nystagmus sometimes develops. *Snell* and others have reported cases in

which this trouble developed in compositors who were accustomed to glance up at their copy without moving their heads, thus making abnormally great demands on their superior recti, and the same explanation probably accounts for the occasional appearance of nystagmus in accountants, and in persons engaged in other occupations, as in the cases reported they were accustomed to turn their eyes far in one direction, usually upward, and to hold them there for shorter or longer intervals quite frequently. But the occupation in which this trouble is most common is a form of mining in which the workman is obliged to do his work with his eyes in a constrained position, which has given it the name of **miner's nystagmus.** *Snell* called attention to the fact that the patients get relief from looking down, and avail themselves so far as they can of positions of the head in which the overtaxed muscles are rested. Miners' nystagmus has been ascribed to the poor light in which the men work, and it is quite possible that this is a contributing factor, but as the trouble is not met with commonly among all miners, who work under similar conditions of illumination, and is confined mainly to those who are obliged to work with their eyes tilted upward, it is probable that the latter is the main factor in its production. The diagnosis is not difficult, provided we have a clear history of the way in which the eyes have been habitually held while the patient has been engaged in his occupation, but otherwise it may be very difficult, especially when the occupation is one in which a tilting of the head instead of the eyes is the usual procedure.

A unilateral nystagmus is to be seen very rarely, and we do not know its cause. Among the causes assigned in some of the reported cases are a foreign body on the cornea, a conjunctivitis, an operation for cataract, and loss of sight.

CHAPTER VII

The Conjunctiva

The conjunctiva forms a sac open along the palpebral margins, which lines the inner surfaces of the lids, is reflected from them to the eyeball, and covers the anterior portion of the sclera to the edge of the cornea. Its epithelium continues over the surface of the latter. It is smooth and lustrous throughout, presents the characteristics of a mucous membrane only in its palpebral portion, where it is closely adherent to the subjacent tissues, and is of a pinkish color, varying to red at the margins of the lids and at the ends of the tarsus, while the transitional and bulbar portions are attached very loosely to the tissues beneath them, and are translucent, almost colorless, so that the white sclera is seen plainly through the latter. No description is adequate to convey to the mind a definite idea of its normal appearance unless it is supplemented by observation, opportunities for which are abundant for all who will avail themselves of them. Within the inner canthus is the **caruncle,** a small, reddish body, an islet of skin provided with glands and hair, which rests partly on a crescentic, vertical fold of the bulbar conjunctiva called the **semilunar fold.** Quite a number of lesions and diseases are common to both the conjunctiva and cornea, so that they may be described with equal propriety under either of these tissues, such as a tumor of the corneoscleral margin, or a phlyctenular keratoconjunctivitis, and other troubles that are considered strictly conjunctival are apt to be associated with corneal complications that form important features in the symptomatology, like the pannus of trachoma, but it seems wiser to describe the diseases of the conjunctiva and cornea separately for the sake of clearness, although in some of these cases the distinction may seem artificial, and in others the most prominent symptom may not lie in the tissue that is the principal seat of the disease.

Before we look at the conjunctiva we have noticed whether the lids are swollen or not, whether any secretion is to be seen on their margins or in the canthi, and whether there is any abnormality observable in the neighboring tissues. The conjunctiva is the first part of the eyeball to receive our attention, and, after observing what is to be seen through the palpebral fissure, we separate the lids

with our fingers, have the patient look down and then up, so as to bring into view all of the cornea, all of the sclera and bulbar conjunctiva that can be exposed, and then evert the lids to examine the palpebral conjunctiva.

To evert the lower lid we place a finger on its skin and draw it down so as to expose the palpebral conjunctiva and the lower transitional fold. To **evert the upper lid** we seize the lashes about the junction of the middle and inner thirds of its margin, draw the lid gently out from the eye until the upper edge of the tarsus alone touches the eyeball, tell the patient to look down, and as he does so press gently on the upper edge of the tarsus with some blunt instrument, like a probe or a finger, when the lid everts instantly. No pain and very little annoyance is felt when this is done properly, and this is appreciated by anyone who has endured the results of clumsy efforts to evert the lids by force without sufficient attention to these details, perhaps at the hands of those who ought to know better. Force is seldom necessary, except when marked pathological changes are present in the lid, but then may be needed. When the lashes are absent the margin of the lid itself is seized at the place mentioned. By this maneuver all parts of the conjunctival sac are brought into view except the upper transitional fold, which seldom needs to be seen, but may be brought to light by seizing the everted lid with a pair, or better two pairs, of forceps and everting it again. A less perfect view may be obtained by slipping an elevator beneath the everted lid and lifting it out from the eyeball.

While we are making this examination we note any deviation from the normal that may be seen in any of the tissues that are visible, and observe whether it is accompanied by signs of inflammation or not. The location of any abnormal condition is perceived at once, as a rule, though sometimes a painstaking inspection with the aid of oblique illumination and a magnifying glass is necessary, but it is not always easy to recognize its exact nature. Ordinarily this inspection of the conjunctiva is brief, but we should train our eyes to follow a routine course in order that nothing may be overlooked.

THE CARUNCLE

The caruncle may be small or large, and has been found double in a few cases. Its hairs are white or colorless, and very fine as a rule, but occasionally they are long, curved, and irritate the con-

junctiva with their points. When the caruncle appears red and angry the first cause we are likely to think of is eyestrain, especially that caused by faulty convergence, but we should never fail to look for the presence of a foreign body, or of an irritating eyelash. Abscess and all manner of tumors have been known to occur in the caruncle, but such cases are rare.

THE SEMILUNAR FOLD

The semilunar fold is implicated more or less in many inflammations of the conjunctiva and may disappear in the body of a pterygium. When it is hypertrophied it may extend a considerable distance toward the cornea. In rare cases we find it inflamed and swollen when none of the surrounding tissues appear to be affected, and then we are apt to find a foreign body beneath it, or an eyelash that is scratching its surface.

CONGENITAL DEFECTS OF THE CONJUNCTIVA

Congenital defects are uncommon. The caruncle may be wanting when the eyeball is abnormally small or absent, the bulbar portion may exhibit an area of thickening in the region of the palpebral fissure, the entire conjunctiva is apt to be absent in cryptophthalmos, and there may be an adhesion of the conjunctival margins of the lids so as to produce a blepharophimosis of greater or less extent. Perhaps dermoid should be considered a congenital defect.

PINGUECULA

Our attention may be arrested by a round, oval, or triangular elevation of a yellowish gray color, looking like a bit of fat, at the inner or outer margin of the cornea in the horizontal meridian of the palpebral fissure. This pinguecula is composed of conjunctiva and subconjunctival tissue that has undergone hyaline degeneration and is of no pathological importance, except that possibly it may become the starting point of a pterygium. It may be mistaken at first for a solitary phlyctenule, especially when a few blood vessels radiate toward it, or for a pericorneal nodule of vernal catarrh, but the absence of all of the subjective symptoms characteristic of these diseases, as well as of all inflammatory symptoms, together with its location and its fatty appearance, usually leave little doubt as to the

diagnosis. If it has been known to exist for some time, or if it remains unchanged for two or three weeks, all possible doubt is dispelled.

SPOTS ON THE CONJUNCTIVA

When we see one or more small, round or oval, reddish, brownish, or black spots in the conjunctiva which are not accompanied by inflammatory symptoms, we should inquire concerning any previous inflammation of the eye, for such spots may be left after the healing of pustules, or of mucous patches on the conjunctiva. In the absence of such a history it may mark an accumulation of nævus cells, and we should determine whether it is increasing in size or not, for it may be the starting point of a sarcoma. It is well to say here that whenever we feel any doubt as to the nature of any conjunctival spot or growth it is best to have it excised and examined under the microscope, for often this is the only way in which a positive diagnosis can be made and malignancy excluded.

PTERYGIUM

When we see a triangular growth which has its base at the internal or the external canthus and its apex at a point on the cornea, and can pass a probe beneath it into a cul de sac at the limbus, both from above and below, we have to deal with a pterygium. The conjunctival portion is called the body, the part on the cornea the head, and that at the limbus the neck. If the body is fleshy and red, while the head has a gray, swollen, gelatinous looking margin, the pterygium is advancing; when growth has stopped the body becomes thin and membranous, and the gelatinous border of the head becomes thinner. The apex progresses toward the center of the cornea, which it rarely passes, and the head remains attached to this tissue by a narrow strip along its middle line. The body blends with the conjunctiva and may obliterate the semilunar fold when it is on the inner side of the eyeball, as it is in the majority of cases. Sometimes two pterygia are present in the same eye, one on the inner, the other on the outer side of the cornea, and four have been known to occur simultaneously in one person.

The **subjective symptoms** caused by pterygium vary. Sufficient astigmatism may be produced to impair vision, yet in many cases there is no complaint of this nature until the head of the pterygium has advanced so far as to interfere mechanically with the sight. The

disfigurement alone is apt to bring the patient to the surgeon. In other cases the conjunctiva is rendered so tense by it as to damage the motility of the eye and cause diplopia when the patient looks in a certain direction, or the growth may so irritate the conjunctiva as to induce catarrhal trouble. Furthermore the involvement of the semilunar fold may so interfere with the lacrimal lake as to cause epiphora.

False Pterygium

A picture somewhat similar to that produced by pterygium sometimes is presented by a fold of conjunctiva that has become adherent to the cornea as the result of a burn, or of some violent inflammation in which the two surfaces were made raw and brought into contact. Such a false pterygium may be found attached to any part of the cornea, coming from any direction, and usually is associated with scars. The differentiation is made easily from the history, its location, the presence of other scars, the absence of a gelatinous looking margin to the head, the fact that it shows no tendency to advance, but has always been of the same size since it was noticed first, and the failure of an attempt to pass a probe beneath its margin, as the fold of conjunctiva is adherent throughout to the surface of the cornea.

TUMORS OF THE CONJUNCTIVA

A tumor of the conjunctiva is recognized readily as such in the great majority of cases, but it is not always easy to tell whether it is benign or malignant. A reddish, yellowish, or grayish tumor at the corneoscleral margin, lying partly in the conjunctiva, partly in the cornea, which is a favorite situation, may be a dermoid, an epithelioma, or a sarcoma. If its surface is rather dry, downy, or hairy, and the history indicates that it was congenital, the diagnosis of **dermoid** is positive; if the surface is smooth and moist it may be any one of the three, with the probabilities in favor of **epithelioma** if it appeared as a small, hard, painless neoplasm in an elderly person, and has grown slowly. A **sarcoma** is more apt to be pigmented, and more likely to overlap rather than to invade the cornea, but occasionally we meet with a pigmented epithelioma that overlaps the cornea, so the differentiation cannot be made in this manner with any certainty. If it was congenital it probably is a dermoid. If it has appeared recently in a young person, the chances

are that it is a sarcoma. When we find a smooth, flat tumor in some other part of the bulbar conjunctiva, where it is freely movable over the sclera, we still have to bear epithelioma in mind if the patient is elderly, but in younger persons it is more likely to be a sarcoma, a fibroma, a dermoid, a lipodermoid, a lipoma, or an osteoma. The presence of some hairs, or the peculiar consistence may render the diagnosis of dermoid, lipoma, or osteoma easy, but in most cases we cannot be sure of its exact nature until after it has been removed. A favorite situation of the dermoid is in the upper outer part, between the external and superior recti, and in the same place we sometimes meet with a lipoma that is connected with the orbital fat by a pedicle, or can feel the tip of a hypertrophied lacrimal gland. The latter can be pushed back beneath the rim of the orbit leaving the conjunctiva unaffected, while we have difficulty in doing this with a dermoid or a lipoma. The conjunctiva moves with the dermoid, and with the lipoma if the latter is in its tissue, otherwise it slides over the surface of the tumor. In rare cases we may see near the inner canthus a dark red, or bluish red growth that is formed of a congeries of blood vessels. This is an **angioma** and is not hard to recognize. A smooth pedunculated tumor covered with conjunctiva is a **polyp,** and will guide us to a lesion caused by a foreign body, or to a wound. A nodulated, pedunculated tumor with a fissured surface that bleeds easily is a **papilloma.** Soft, irregular granulations, that bleed readily and are not covered by conjunctiva, sometimes spring up about a wound and indicate its presence.

Cysts of the Conjunctiva

A cyst is recognized at once when we see the surface of the conjunctiva elevated by a little sac of clear or yellow fluid. A cyst with clear, watery contents, which is situated superficially in the bulbar conjunctiva, probably originated from a dilated lymphatic. Quite often a number of small, transparent beads of this nature are to be seen between the cornea and the canthus in the region of the palpebral fissure; these are known as **lymphectasiæ** and cause little or no trouble, except sometimes to worry the patient. Other cysts result from pathological changes in the epithelium, or the agglutination of little folds of the conjunctiva; such are to be seen rarely in the head and neck of a pterygium. Others are of traumatic origin and have followed wounds; some familiar examples of

these are the cystoid cicatrix sometimes seen after an iridectomy or a cataract extraction, and the cushion produced by trephining the sclera. When the wound has pierced the conjunctiva alone we are unable, as a rule, to determine by clinical inspection whether the cyst has been caused by the agglutination of folds, by the transplantation of epithelial cells into the subconjunctival tissue, or by the presence of a foreign body. Similar cysts are found on the palpebral conjunctiva, as well as others caused by an occlusion of the ducts of Krause's glands. A bluish, cystlike spot in the conjunctiva over a Meibomian gland marks the presence of a chalazion.

The possibility of a **cysticercus** should be borne in mind when we find a large cyst in the conjunctiva, though this parasite seldom is found anywhere in the eye in this country. While the cyst is still transparent the head of the cysticercus may be seen within it; later, when its surface has become inflamed and opaque, it should be incised and searched carefully.

SYMBLEPHARON

Quite frequently we find an agglutination of more or less of the palpebral to the bulbar conjunctiva. This is called a symblepharon. The usual cause is a burn which denuded the two surfaces so that they became adherent while they lay in contact afterward, but the same result sometimes is produced by a wound, by a purulent or membranous conjunctivitis, by trachoma, and by pemphigus.

XEROSIS

An abnormally dry condition of the conjunctiva, or of the conjunctiva and cornea, which has been produced by prolonged exposure to the air, by cicatrization that has followed local inflammation, or by certain depleted states of the body, is called xerosis. It may be partial in lagophthalmos and in ectropion, but, as a rule, the entire surface is involved. It is met with in two forms, a milder and curable one in which the epithelium alone is affected, and an incurable one in which the conjunctiva has been changed into cicatricial tissue. The epithelial form appears together with hemeralopia in some cases of starvation, chronic alcoholism, and certain chronic diseases of the liver, as well as in desperate cases of cholera, cholera infantum, dysentery, typhoid fever, scarlet fever, puerperal fever, and congenital syphilis. The worst form is seen after the

conjunctiva has been destroyed by cicatrization following severe burns, trachoma, pemphigus, or the worst cases of membranous conjunctivitis, when the membrane cannot be moistened by the lacrimal fluid that flows over it in the cases in which the function of the lacrimal gland is preserved to a greater or less extent. The subjective symptoms are those of an intense feeling of dryness.

SUBCONJUNCTIVAL HEMORRHAGE

An effusion of blood beneath the conjunctiva is at first of an even, bright red color, and is distinguished easily from an inflammatory redness by the absence of vessels within it, and its sharp delimitation. During absorption its boundaries become more diffuse, while the color becomes darker, and then changes to a variety of hues spread diffusely over an area on the surface of the eyeball proportionate to the size of the hemorrhage.

The commonest cause of such an effusion is **inflammation.** Numerous little extravasations are to be seen in the upper, less often in the lower part of the bulbar conjunctiva in many cases of acute conjunctivitis. The next most common cause is **traumatism,** which may be either direct or indirect. A wound of the conjunctiva, whether accidental or operative, is followed usually by an ecchymosis. After a contusion it forms a part of the picture of a "black eye." An extensive ecchymosis that follows a blow on the head is symptomatic of a fracture of the wall of the orbit, or of the base of the skull; it may appear soon after the injury, or not until after the lapse of some time, and one that appears late on the temporal side is rather suggestive of a fissure that extends into the orbit through the sphenoid bone. When caused by a contusion, a fracture, or a severe compression of the thorax and abdomen, it is associated usually with subdermal ecchymoses in the lids.

Hemorrhages occur frequently in children during paroxysms of whooping cough, less often in older persons during coughing, sneezing, vomiting, violent exertions, straining at stool, or labor pains. **Spontaneous** hemorrhages take place, often at night while the patient is asleep, in persons who have brittle blood vessels, when they are apt to cause considerable fright. Such patients ordinarily are elderly and have arteriosclerosis, but sometimes they are young, and then it will commonly be found that the walls of their vessels have been weakened by some such disease as nephritis, syphilis,

diabetes, scurvy, malaria, or purpura. The integrity of the eye is not threatened by a spontaneous subconjunctival ecchymosis, but as it indicates an abnormal condition of the blood vessels it should call for a thorough investigation of the organism; it may be a forerunner of an attack of apoplexy, or a symptom of some serious general disease.

EMPHYSEMA OF THE CONJUNCTIVA

When the conjunctiva is lifted up from the eyeball so as to form a tense swelling in which a sense of crepitation is communicated to the fingers when they are pressed upon it, air has entered beneath it. This condition frequently accompanies emphysema of the lids, and is due to the same cause. The only other condition for which it may be mistaken is chemosis, from which it is differentiated by the feeling of crepitation.

CHEMOSIS

Very often we see a clear or opalescent puffiness of the bulbar conjunctiva, or a denser, yellowish thickening that surrounds the cornea like a wall, sometimes overlaps it, and may involve the transitional folds, but in which no crepitation can be felt. This is chemosis, which is due to a distention of the subconjunctival tissue with serum, often together with an inflammatory exudate. The swollen conjunctiva is crowded for space and a fold of it may protrude between the lids. The color changes to red in severe inflammation, while the pressure of the swollen upper lid tends to drive the œdema downward, when the protrusion between the lids may be that of a large smooth red roll. Should the surface of this protruding fold not be kept sufficiently moist by the secretion that usually flows abundantly over it, a part may become dry from exposure to the air, and then ulcerate or necrose.

Chemosis is a striking symptom, and while it may or may not indicate a serious lesion, its presence calls upon us to ascertain at once whether its cause is an obstruction to the outflow of blood and lymph due to inflammation or something else, or a morbid condition of the blood. It is a very common early symptom of many inflammations, not only of the conjunctiva itself, but also of various tissues of the eyeball and of the adjacent parts, it occurs in certain general diseases, and it may also be produced by drugs. We meet with it after both trivial and severe traumatisms to the

conjunctiva, in both mild and severe cases of acute conjunctivitis, and it may be the first sign to warn us of the presence of infection after such an operation as a cataract extraction. It is common in all purulent infections of any of the tissues of the eye, as well as in other inflammations that are not purulent, such as iritis, iridocyclitis, and glaucoma. We are apt to see it in connection with an inflammation of the lids like a hordeolum, an acute dacryocystitis, a cellulitis of the orbit, a periostitis of the margin of the orbit, or an inflammation of any of the orbital tissues. Associated with œdema of the lids it is not uncommon in frontal sinusitis and ethmoiditis, and is frequently present in cases of facial erysipelas, and of the eruption on the face caused by poison ivy.

Less often it occurs when no signs of inflammation are present. When we see an exophthalmos without inflammation, but with chemosis, we suspect that a retrobulbar tumor impedes the outflow of blood from the orbit. Chemosis seems to be caused occasionally by anæmia and chlorosis; sometimes it is a symptom of nephritis; in these cases it may come and go, though this is not the rule. Another fugacious chemosis has been ascribed to atmospheric changes. A regularly recurring chemosis in women may be connected with menstruation, and *Roemer* has observed it in repeated attacks of migraine. It has been known to occur in urticaria and in trigeminal neuralgia. A slight localized œdema may be observed sometimes over a paralyzed muscle. A chemosis has been known to follow the internal administration of quinine, or of potassic iodide, and it is ordinarily produced by the instillation of a solution of dionin into the conjunctival sac. The cushion formed by the escape of aqueous beneath the conjunctiva through an aperture in the wall of the globe at the corneoscleral margin, made perhaps by a trephine, is called sometimes a filtration chemosis. Yet, in spite of all these possible causes, we occasionally meet with a case for which we are not able to account.

REDNESS OF THE EYEBALL

Two or three blood vessels that run separately in the bulbar conjunctiva, and have no morbid symptoms associated with them, are of no importance; they are normal to the individual, who will not feel grateful for attempts to remove them by astringents that simply irritate the eye, or for being induced to wear glasses that

neither conduce to his comfort, nor have any effect upon them. But when we see either a general or a localized redness that is caused by the injection of quite a number of neighboring vessels, we must accept it as showing the presence of trouble somewhere in or about the eye. We should note the location and character of such a redness, as well as take into account the history and the accompanying symptoms, for it may be a sign of the presence of a foreign body on the conjunctiva or cornea, of conjunctivitis, keratitis, scleritis, or glaucoma, of an inflammation of the lid, of the lacrimal organs, of the accessory sinuses, or of the retrobulbar tissues. In some of these cases the redness is caused by a congestion of the conjunctival vessels, in others of the episcleral, so the first thing for us to determine is whether the enlarged vessels are in the conjunctiva, in the episclera, or in both. When the redness increases as we pass from the cornea toward the transitional folds and accompanies the conjunctiva when this is moved back and forth over the surface of the globe, the congestion is conjunctival; when it increases in depth toward the cornea and remains in place while the conjunctiva is moved over it, the injection is episcleral. In a great many cases we find both conditions to be present.

Many conjunctival troubles give rise to a feeling of discomfort, heat, burning, itching, scratching, or pain, but some do not, and this is one of the reasons why a routine examination should be made in every case that comes to our attention. Frequently we find faults in the palpebral conjunctiva when the bulbar appears to be perfectly well, and the reverse is true in some cases. The palpebral conjunctiva may be anæmic, hyperæmic, or inflamed, when the patient complains of an indefinite feeling of irritation of the eye.

Anæmia of the Conjunctiva

If the palpebral conjunctiva of the lower lid is pale it is well to evert the lower lip to see if its color confirms the suggestion of a general anæmia. I am convinced that anæmia itself gives rise to subjective sensations of irritation of the eyes in many cases, for otherwise it is hard to explain why the annoyance persists as long as the anæmia, in spite of a careful correction of all refractive and muscular errors and of any other abnormal conditions that may be present, to pass away when the general condition improves. This condition seems to be met with most frequently in elderly persons, in whom it is apt to be obstinate and troublesome.

Hyperæmia of the Conjunctiva

When we find the palpebral conjunctiva to be too red, but not looking velvety, and with no discharge beyond possibly a little dry bit in the inner canthus in the morning, perhaps with a moderate amount of lacrimation, we pronounce it hyperæmic. Such a condition may be produced by many causes, some of which are hard to detect. We think first of refractive and muscular errors, which furnish a large contingent, but nasal troubles account for many cases, and exposure to wind, smoke, and dust is a prolific source. Sometimes it is caused by lack of sleep, it accompanies hyperæmia of the margins of the lids as a rule, and it may be due to such a general disease as the uric acid diathesis, or paralysis of the cervical portion of the sympathetic nerve. Toxic causes must not be forgotten. It is well known to result from abuse of alcohol and tobacco, but it may be caused by a slight indulgence in either. A single cigar, or a single glass of liquor, has been known to produce an acute hyperæmia in rare cases, and habitual indulgence in small quantities may excite a similar chronic condition. The vessels in a distinctly localized hyperæmia may diverge toward a foreign body, or may conceal the latter in their midst.

CHRONIC CONJUNCTIVITIS

The dividing line between a well marked hyperæmia and a mild chronic conjunctivitis is not well defined, unless we assume the least demonstrable trace of secretion to mark the transition, but sometimes we find such a trace when none of the other symptoms seem to justify us in pronouncing the condition anything more than hyperæmia. On the other hand we may not be able to find any secretion even when the slightly roughened appearance of the palpebral conjunctiva may lead us to say that a chronic conjunctivitis is present. The one condition passes over into the other, and all of the causes of conjunctival hyperæmia may excite conjunctivitis. The **cardinal symptoms** are a chronic redness and swelling of the palpebral conjunctiva, which may vary from a very slight redness and roughening, to a red, uneven surface that tends to appear velvety in the transitional folds, and a sticky mucus that dries on the margins of the lids and may be sufficient to glue them together during sleep. In most cases the bulbar conjunctiva is af-

fected slightly if at all, but sometimes it is injected, and then the eyes appear to be reddish and irritable, while the patient cannot look at a bright light without discomfort and lacrimation. The caruncles and the margins of the lids often are red and prominent.

This is distinctly an **occupational** disease in many callings, in which the eyes are exposed continually to wind, dust, smoke or heat; it is common and very obstinate among farmers, coachmen, automobilists, cooks, and foundrymen. It may be produced by such local causes as a pterygium, a chalazion, the presence of concretions beneath or in the conjunctiva, disease of the lacrimal duct, and inflammation of the margins of the lids, or it may depend on nasal troubles, or be symptomatic of the uric acid diathesis. In all of these cases the condition is intractable until the cause has been removed. It may be, though it is not very commonly, a sequel to acute conjunctivitis.

The bacteriological findings vary and are of little importance, except when the diplobacillus of Morax-Axenfeld is present, which produces a characteristic variety of its own.

Diplobacillus Conjunctivitis

When the margins of the lids are red only at the canthi, and this redness is most pronounced at the inner one, where the caruncle, with perhaps the adjacent skin, is covered by a small quantity of yellowish, tenacious mucus, the presence of the diplobacillus of Morax-Axenfeld is probable, and we have reason to feel very confident of the presence of this microorganism if we find very slight changes in the palpebral conjunctiva, often amounting to little more than redness near the transitional folds, and none in the bulbar except redness near the canthi. The bacilli are short, stout, usually in pairs, and may be found in large numbers in a smear taken from the mucus that covers the caruncle. They withstand drying for a long time, especially when they are in dry secretion, and when they have once settled on the human conjunctiva they set up this mild, chronic inflammation, which is of indefinite length unless properly treated.

FOREIGN BODY ON THE CONJUNCTIVA OR CORNEA

The symptoms excited by the presence of a minute foreign body on the surface of the eye, pain, photophobia, lacrimation, and a

reddened eyeball, are well known, for there are few of us who have not suffered from them personally. As a rule the patient comes to us with the diagnosis made correctly, and we are able to see the foreign substance caught on the surface of the cornea, or to find it on the tarsal surface of the upper lid. A favorite place for lodgment in the latter case is the subtarsal sulcus, which runs horizontally about 3 mm. above the margin of the lid. If we do not see it at once it is best for us to employ oblique illumination and a magnifying glass, or, better, a binocular loupe, to aid us in making a thorough investigation of the entire surface of the cornea and of the upper palpebral conjunctiva. We are likely to get help from the instillation of a drop of a solution of fluorescin, which usually stains green the slightest erosion of the corneal epithelium, but for some unknown reason it sometimes fails to do this, even when the erosion is plainly visible, so it is not absolutely reliable. In spite of this fact the assistance it affords is so great that it is well to use it whenever we suspect, but cannot see an abrasion of the epithelium of the cornea. A swollen, reddened semilunar fold always invites exploration beneath it. A group of distended conjunctival vessels that converge toward a certain place in the periphery of the cornea is quite apt to guide us to a minute particle entangled in the corneal epithelium, and a distinctly localized congeries of vessels in the palpebral conjunctiva may conceal a similar one. A minute bit of sawdust, glass, quartz, or other material that is either colorless, or of a pale yellow tint which resembles the basal color of the palpebral conjunctiva, may be extremely difficult to find; sometimes such a substance can be located by a small collection of whitish mucus at a certain point on the surface of the membrane.

Not infrequently the sensation persists after a foreign body has escaped, or has been removed, but in such cases an **abrasion** of the corneal epithelium can be detected almost always by a careful inspection. It is possible for the subjective sensations to be misleading, and to indicate not the presence of a foreign body, but the **onset** of an **acute catarrhal conjunctivitis,** but such a diagnosis is not to be made except when the conjunctival hyperæmia is general, rather than localized at any point, no little circumscribed collection of mucus can be found, and no trace of a foreign body, or the slightest lesion of the corneal epithelium has been detected after a thorough, painstaking search.

ACUTE CATARRHAL CONJUNCTIVITIS

When a patient complains of a sudden attack of pain, lacrimation, and photophobia, soon followed by a watery discharge from the eye, we think at once of an acute catarrhal conjunctivitis, but should not hazard the diagnosis on these symptoms alone. The pain may vary from a vague feeling of discomfort to severe, and sometimes is like that produced by the presence of a foreign body. The photophobia may be intense, or absent, but ordinarily is quite moderate. The clinical symptoms vary with the severity of the attack. Usually the margins of the lids are reddened, often are a little œdematous, the palpebral conjunctiva is red and smooth, perhaps showing separate enlarged blood vessels if the case is seen early, or the attack is mild, while the transitional fold is very red and somewhat swollen. The bulbar conjunctiva may be unaffected, reddened, or chemotic in mild cases, in severe ones the redness and chemosis are quite marked.

Sometimes a general practitioner is heard to say that he does not know much about the eyes, but he knows conjunctivitis when he sees it. We may feel a little skeptical, for it is hard to find an expert who does not make a mistake occasionally, especially when the small amount of secretion is washed away to a great extent by an abundant lacrimation and the bulbar conjunctiva is injected, for such a clinical picture may be caused not only by the onset of an acute catarrhal conjunctivitis, or the presence of a small foreign body on the surface of the eye, but also by keratitis, iritis, and glaucoma. The presence of a foreign body may be excluded by inspection. The diagnosis of keratitis is easy if the lesion in the cornea is large and in plain view; so is that of iritis if the pupil is small and irregular, if the redness of the eyeball increases in depth toward the cornea, and if the iris looks muddy; so is that of glaucoma if the pupil is widely dilated, the cornea hazy, anæsthetic, and surrounded by a zone of tortuous, purplish vessels, while the pain is agonizing and the eyeball hard; but these characteristic symptoms are not always well defined in any of these diseases, and the nature of the trouble may not be so evident. First we must determine whether the redness of the eye is due to congestion of the conjunctival, or of the episcleral vessels, or of both, in the way already described. If the injection is purely conjunctival and the pupil is normal all of these inflammations have been excluded except conjunctivitis, with

the possible exception of a very early stage of iritis, for in a minority of cases the latter may set in with a conjunctival injection, a pupil that is normal or slightly dilated, and no noticeable change in the iris, when, as *Roemer* remarks, "the diagnosis is not made." But the number of errors of this nature can be much reduced by careful attention to all of these symptomatic details in every case:—The nature of the pain, whether it is worse at night or not, as pain that wakes the patient at night is quite suggestive of iritis in such a case, careful inspection to see whether the episcleral vessels are involved in the least, the appearance of the iris as compared with that of the other eye, and the size and behavior of the pupils. Mydriatics and cycloplegics are of no use in the treatment of conjunctivitis, yet when any of these diagnostic points seems a little suspicious, the instillation of a drop of atropine, or of homatropine, may aid us to reach a conclusion. Occasionally we are gratified by the statement of the patient a few hours later that the drop has given relief, an almost certain sign that the iris is involved, but we must be circumspect in employing this means in elderly patients.

Later the **clinical picture** of an acute catarrhal conjunctivitis is characteristic, though the degrees of all of the symptoms correspond to the severity of the attack. The margins of the lids may be slightly œdematous, or the lids as a whole may be red, swollen, and closed over the eye. The skin along their margins is apt to be excoriated by the discharge, by which the lashes are matted together more or less. Quantities of secretion may be seen exuding from between the lids as mucopus or pus, or floating about when the lower lid is drawn down. The palpebral conjunctiva is red, swollen, and less translucent than normal; its surface may be smooth and glistening, or velvety, or resembling granulations in some cases. The transitional fold bulges forward when the lower lid is everted, and the redness from this spreads into the bulbar conjunctiva, growing lighter toward the cornea. Chemosis may or may not be present. If we have the patient look down while we lift the upper lid we can usually see little red spots which have been caused by subconjunctival hemorrhages; these are not apt to occur below the cornea except in severe attacks.

Most cases of acute catarrhal conjunctivitis are excited by **microorganisms,** some of which are accustomed to give rise to clinical pictures that are more or less characteristic, but these are simulated sometimes pretty accurately by nearly all of the others. The micro-

organisms most often responsible for an attack of this disease, at least in this part of the world, are the Koch-Weeks bacillus and the pneumococcus; others that are considerably less common are the staphylococcus, the streptococcus, the influenza bacillus, the pneumobacillus, the colon bacillus, and perhaps a few more. We must add the diphtheria bacillus, as this has been demonstrated in mild catarrhal attacks, though ordinarily it excites a typical membranous conjunctivitis, so inflammations caused by it will be considered under that heading. The gonococcus gives rise to a violent purulent form of conjunctivitis in adults, and a less violent form in infants, both of which will be considered separately, and is so virulent that we never hear of it causing mild symptoms. Many years ago a large number of smears taken from a case of mild conjunctivitis that recovered in three days, were loaded with microorganisms which were pronounced by one of the best bacteriologists of the time in New York City to be gonococci. It has always been a matter of regret that no cultures were made, but this procedure was little known at the time, for although the morphological resemblance to gonococci was perfect, it is probable that the bacteriologist was mistaken, and that some other unknown microorganisms were present. If he was correct this is the only case of which I have been able to find record in which the gonococcus excited a mild, catarrhal conjunctivitis. With the exception of this one all of the microorganisms mentioned may excite a simple catarrhal inflammation in one case, a purulent in another, and a membranous in a third, so it is never safe to omit a bacteriological examination of secretion taken from the conjunctival sac, not from the edges of the lids, even though it is very rare for some of them to excite one of the severer forms of inflammation. A few points that may enable us to conjecture whether the pneumococcus or the Koch-Weeks bacillus is the ætiological agent, but not to diagnose it, are these:—A pneumococcal conjunctivitis usually begins suddenly and ends by crisis, much like a lobar pneumonia, and generally recovery is complete in about ten days, no matter what course it may run. A conjunctivitis caused by the Koch-Weeks bacillus lasts two or three weeks, the average photophobia seems to be more marked, the patients are more apt to complain of pain in the orbit and upper maxilla, and a swelling of the preauricular gland is more common.

Acute catarrhal conjunctivitis of microbic origin usually is bilateral, though one eye is likely to be affected before the other, and

is a common accompaniment of all catarrhal affections of the upper respiratory passages, especially of a cold in the head. It is common in various acute infectious diseases, and forms a prominent symptom in measles, in which it generally runs a mild course, though sometimes it assumes the membranous form, and the swelling of the preauricular gland is not marked, as a rule. Rarely, and mostly among the employees of slaughter houses, we meet with cases caused by inoculation of the conjunctiva with dead animal matter.

All cases are not microbic, for a very similar inflammation may be caused by a mechanical or chemical **traumatism.** One eye alone may be affected when the cause is a blow, the lodgment of a foreign body, or the entrance of an irritant solution or drug, but usually both eyes are inflamed when the condition is the result of exposure to a bright light, to heat, or to the invisible rays of the spectrum. The symptoms in these last cases resemble closely those produced by very superficial burns, a sudden attack of severe pain with much photophobia, redness, and lacrimation, but not a great deal of swelling or discharge.

The diagnosis of acute catarrhal conjunctivitis cannot be said to be complete until its ætiology has been determined, and all of the other forms of acute conjunctivitis about to be described have been excluded by the absence of their characteristic features.

SQUIRREL PLAGUE CONJUNCTIVITIS

In 1914 *Vail* described a purulent conjunctivitis characterized by ulcers of the conjunctiva, a tender, swollen preauricular gland, an extension of the inflammation to the lacrimal sac, and the formation of pustules between the eye and the ear, which he believed to be the first case on record of a squirrel plague conjunctivitis. Experiments on guinea pigs with scrapings from the ulcers induced a similar disease characterized by a bacillus believed to be identical with one pronounced by *McCoy* and *Chapin* to be the agent of squirrel plague.

OPHTHALMIA NODOSA

When an acute catarrhal conjunctivitis is complicated by the presence of grayish red, or yellow nodules scattered about in the bulbar conjunctiva, we inquire immediately whether the patient has been struck in the eye with a caterpillar, or a hairy plant, as the hairs of certain species of caterpillars are quite apt to produce this

condition, and those of certain plants are said to have done likewise. Sometimes we can see a hair protruding from a nodule, or we may find one within it, which establishes the diagnosis of ophthalmia nodosa and excludes the possibility of tuberculous nodes. If these cases are neglected the hairs are apt to penetrate through the cornea and sclera into the eyeball, where they will set up a serious inflammation of the iris and choroid. When we can obtain no such history as the one indicated above, and no hairs can be seen projecting from the nodes, one of the nodules should be excised and submitted to a microscopic examination.

GONORRHEAL CONJUNCTIVITIS

The symptoms excited by gonococci in the conjunctivæ of adults and of infants differ so much that they have to be considered separately.

Gonorrheal Conjunctivitis of Adults

When the lids of an adult become so swollen, red, and tense within a few hours that the patient is unable to open them, while a thin reddish or yellowish fluid wells forth from the palpebral fissure, and he complains that the eye is very painful and exquisitely tender, we suspect gonorrhea at once, for this violent onset is so characteristic that we seldom think of other possible causes until gonococci have been proved to be absent.

We separate the lids with an elevator, taking great care not to touch the cornea, and then we see that the bulbar conjunctiva is red, very chemotic, surrounding and perhaps overlapping the cornea with a firm, doughy wall, and that it contains hemorrhages of various sizes and shapes. The cornea must be inspected carefully, for the least haziness of its tissue, or abrasion of its epithelium, gives a bad prognosis, which is made worse by the presence of an ulcer. When the lower lid is drawn down the transitional fold springs forward as a thick, red roll, often with gray patches upon it, while the palpebral conjunctiva is very thick, red, and velvety. As a rule we do not evert the upper lid, because it is so swollen that it cannot be everted without more violence than is justified by what we are likely to find. In the rare cases in which the history proves misleading and no gonococci are to be found, we evert the lid as soon as we receive the negative report and investigate the upper transitional fold in search of a foreign body, like a beard of grain, that

may have lodged therein several days before and have set up a violent purulent inflammation, if no other cause is apparent. The preauricular gland is almost always swollen and painful in gonorrheal ophthalmia, and the patient has a certain amount of fever.

The inflammation usually appears first in one eye, but soon extends to the other, unless measures are taken to prevent its infection. It reaches its acme in about ten days and then subsides gradually. Recovery may be complete in a month or two, but sometimes a chronic condition is left, which is characterized by redness and thickness of the palpebral conjunctiva with hypertrophied papillæ.

The prognosis is bad. An eye seldom escapes damage and many are blinded. The most serious and most frequent complication is an involvement of the cornea in which the nutrition of that tissue is impaired, with the formation of ulcers that are prone to perforate. Less often the inflammation extends without perforation to the iris, the ciliary body, and the deeper structures, with an equally bad result, so far as vision is concerned.

A purulent conjunctivitis is ***not necessarily gonorrheal.*** It may be excited by any of the microorganisms that cause acute catarrhal conjunctivitis, but then the onset usually has been more gradual. If the patient has had a catarrhal conjunctivitis for two or three days which has increased in severity until it has become violent and purulent, the probability is that we shall not find gonococci. The onset is sudden when the conjunctivitis is caused by pneumococci, but ordinarily the symptoms are not so violent, and the eyeball is less painful and tender. Still the resemblance is so close in many instances that nothing but the bacteriological examination can establish the diagnosis. The prognosis of a purulent conjunctivitis is better when gonococci are not present, but we must not consider the eye out of danger when these microorganisms have been excluded, for a purulent inflammation of the conjunctiva often is a serious matter, no matter what the exciting agent may be.

Metastatic Gonorrheal Conjunctivitis

A simple acute catarrhal conjunctivitis that appears in a patient who is suffering from gonorrhea or gleet, with no gonococci in the discharge, no other microorganisms present in sufficient quantity to account for the disturbance, and no other known cause, is quite apt to be metastatic. We find the redness to be confined mainly to the transitional folds. This form is perfectly benign, but it is apt to recur with each attack of the fundamental disease.

Gonorrheal Conjunctivitis of Infants

When we speak of ophthalmia neonatorum we may be understood to refer to this, the worst variety of **babies' sore eyes,** or to a group of inflammations met with in the eyes of infants, in all of which an inflammation develops in the conjunctiva a few days after birth. If it appears within two or three days the inoculation of the eyes probably took place during parturition, but when the onset is delayed for several days it is more likely that the infection occurred later. Both eyes are attacked simultaneously, as a rule, though the infection may appear in the second one after an interval of twenty-four hours or more, but the disease very rarely is confined to one eye alone. Whenever we see that a baby has even a very slight discharge from the eyes we should take a smear and examine it as quickly as possible, for the gonococcus does not make such a fulminating attack on the conjunctiva of a baby as it does on that of an adult, and there is no way in which we can determine immediately whether the eyes are in serious danger or not, aside from the bacteriological examination. The pneumococcus, Koch-Weeks bacillus, and colon bacillus may excite a purulent inflammation, while malignant attacks have been caused by the diphtheria bacillus, and the diplococcus intracellularis. If no microorganisms are found in the smear we should inquire whether Crede's procedure was followed, as this sometimes causes a rather sharp catarrhal attack, occasionally accompanied by bleeding.

The **first sign** noticed by an observant nurse is a little discharge, or a gluing of the lids together, but within a few hours the lids become red and so tensely swollen that they are difficult to separate, while the discharge accumulates behind them. The physician must be careful for his own eyes when he attempts to make an examination during this stage, for the watery or yellowish fluid may spirt to a considerable height as soon as he parts the lids. It is well for him to wear protective goggles when he is handling any of these very contagious cases, and also to guard against this sudden spirt when he opens the eye by laying a bit of wet cotton over the lids to catch the discharge when he parts them first. This done he may remove the cotton and lift the upper lid with an elevator, taking care not to touch the cornea. At first the palpebral conjunctiva is red, smooth and glistening, the bulbar unaffected, reddened, or chemotic. The last is a bad sign when seen in this stage, for it indi-

cates that the infection is virulent. After a few days the discharge changes to a profuse quantity of yellow pus, the lids become less swollen and tense, the palpebral conjunctiva velvety. This stage may last for several weeks. The velvety palpebral conjunctiva gradually becomes covered with elevations caused by the hypertrophy of the papillæ, until it resembles granulation tissue, the bulbar conjunctiva becomes thickened, the discharge lessens gradually, changes to mucopurulent, then to watery, finally stops, and the conjunctiva slowly regains its normal condition, except perhaps for some cicatricial changes.

The prognosis is better than that of gonorrheal conjunctivitis of adults, yet it is very grave, as the records of all institutions for the blind can testify. The onset of the disease should be guarded against carefully, not only through cautious antisepsis during labor, and the subsequent protection of the eyes from any possible infection, but also by cleansing the eyes with close attention to details. When the genital passages of the mother are known or suspected to be infected with gonorrhea the instillation of a drop of a two per cent. solution of silver nitrate into each conjunctival sac, as recommended by Crede, should never be omitted, even though the severe reaction that follows once in a while may render it undesirable for routine use when there is no warrant for suspicion of such disease in the mother, but at least a one per cent. solution should always be employed. In addition to this the eyes should have their first bath from a basin of clean water, and none of the water used to bathe the body should be allowed to come in contact with the eyes. In spite of all precautions a conjunctivitis will appear sometimes in a baby's eyes, and then stringent measures should be taken at once, holding the infection to be gonorrheal until it has been proved otherwise. No matter how slight the symptoms, the danger is great, and it is far better to take unnecessary precautions many times than to have a single child become blind through their neglect.

MEMBRANOUS CONJUNCTIVITIS

A wound of the conjunctiva may become covered with an exudate of fibrin that forms a membrane beneath which healing takes place, and the same is true of burns with hot metals, acids, or alkalies, but in these cases the history helps us to an immediate diagnosis, whether the membrane be superficial and easily removed, or deep and ad-

herent. We do not refer to such cases when we speak of membranous conjunctivitis, but rather to an inflammation caused by microorganisms and characterized by the formation of a membrane that may lie upon, or be incorporated with the conjunctiva. Cases of this nature were divided into those of croupous, and of diphtheritic conjunctivitis in the olden time, when diagnosis had to be drawn wholly from clinical appearances, without the aids of the microscope and experimental research. The distinction was that a false membrane which could be detached easily, and left a raw, bleeding surface on the conjunctiva was **croupous,** while one that could not be detached easily, but involved the deeper layers of the conjunctiva was **diphtheritic.** This distinction is still useful as a means by which to indicate the milder and graver forms of membranous conjunctivitis, if we will bear in mind that the only difference between them is one of severity, not one that necessarily indicates the active agent. The microscope has revealed the diphtheria bacillus in many croupous cases, and demonstrated its absence in others of the diphtheritic variety, while experiments have proved that a croupous or a diphtheritic inflammation can be excited at will by the application of certain strengths of ammonia or jequirity to the conjunctiva. All manner of intermediate grades also are to be met with between the typical conditions of croupous and diphtheritic conjunctivitis, so it can hardly be doubted that we have to deal, not with two, but with one form of inflammation, in which the symptoms vary with the virulence of the morbid agents, the local condition of the conjunctiva, and the susceptibility of the body as a whole.

The majority of cases are met with in ill nourished, badly run down children, often when they are convalescent from some such acute infectious disease as measles, or scarlet fever, or else during the course of an attack of diphtheria. The **active agent** may be any of those capable of exciting an acute catarrhal conjunctivitis, but those most often met with are the diphtheria bacillus, the streptococcus, and the gonococcus. It is notable that while the streptococcus rarely appears in the catarrhal conjunctivitis which accompanies measles and other infectious diseases, it is usually present in force when the inflammation changes to the membranous form.

In the mild, or **croupous,** cases there is first an attack of acute catarrhal conjunctivitis, then the lids become more swollen, though they remain soft, and a grayish, rather translucent membrane of coagulated fibrin forms over the inner surface of the lids, starting

from the transitional folds. This can be removed easily, and leaves a raw surface that tends to bleed, but it soon reforms. In the mildest cases the upper lid can be everted readily, the bulbar conjunctiva is not involved, the cornea seldom is implicated, and the false membrane disappears in about two weeks. The diagnostic acumen of our fathers is shown by the fact that although this mild, croupous form sometimes is excited by the diphtheria bacillus, in the great majority of cases the active agent is some other microorganism, while this bacillus abounds in most of the bad cases they called diphtheritic.

In severer cases the swelling of the lids increases, they become firmer and harder to evert, the membrane becomes grayer, less translucent and less easily stripped off; the conjunctiva bleeds more and more readily at the slightest provocation, and the bulbar conjunctiva shows signs of inflammation. These may be called the intermediate forms, which are hard to classify as either croupous or diphtheritic, that is as mild or very bad.

In a very severe case of membranous conjunctivitis, one that we may call **diphtheritic,** the initial symptoms are much like those of gonorrheal ophthalmia, except that the pain is worse, and that there is no great amount of discharge. The lids are red, swollen, feel hard and boardlike, and the upper one cannot be everted. When we separate them a small quantity of thin, turbid fluid tinged with blood wells up; after this has been wiped away we see that the bulbar conjunctiva is chemotic, of a livid, yellowish color, and we find that the palpebral conjunctiva presents a dirty gray, opaque membrane which may be continuous, or appear only in patches. This membrane is hard to remove, and when torn away does not leave a bleeding surface, because the conjunctiva has been deprived of blood by the coagulation of an exudate which has compressed the vessels, a condition that is apt to pass into gangrene, which may invade the tarsus, or other parts of the lid and rapidly destroy the eye. The symptoms begin to change at the end of a week or ten days. The lids become softer, a purulent discharge appears, the membrane melts away and leaves the surface red, raw, and bleeding very easily. Finally, cicatrization takes place, and this may cause a great deformity of the lids, or even a displacement of the eyeball, if it has survived.

This malignant form suggests diphtheria, and this diagnosis probably is correct if the patient presents evidences of diphtheria else-

where, but it can be made positive only by a bacteriological examination, not simply of a smear, but of a culture as well, because the conjunctiva is the normal habitat of the xerosis bacillus, which looks exactly like that of diphtheria. A culture brings out identifying characteristics in a few hours, and absolute certainty is to be attained through proof that the bacilli found are able to form antitoxin. Next to the diphtheria bacillus the streptococcus probably is the microorganism most often responsible for these very grave cases. Sometimes such a membrane is to be seen in a very bad case of gonorrheal ophthalmia, and other agents are said to be guilty occasionally.

The diagnosis of membranous conjunctivitis as such is easy. The only condition in which an error would seem to be possible is the very rare acute pemphigus, in which the characteristic violent symptoms are lacking.

VERNAL CATARRH

When a patient complains of an annually recurring irritation of his eyes with photophobia and itching, alternating from time to time with a feeling of pressure and stinging pain like that produced by a foreign body, which begins in the spring, continues during the summer, and passes away gradually as the weather becomes cool, he gives a history characteristic of the rather rare disease vernal catarrh. The secretion is slight at first, when the attack, or better the exacerbation, sets in, and increases gradually until the ordinary picture of catarrhal inflammation is presented. Sometimes the discharge is free, but it is to be found characteristically in the form of long, tenacious, transparent threads that may be removed from the transitional folds by irrigation, or with forceps.

The upper lid droops so as to give a sleepy expression to the face, and when we evert it two things attract attention immediately—the peculiar, pale, bluish red, milky color of the conjunctiva, and the presence in its tarsal portion of roundish, flat, hard excrescences that may be separate, but usually are packed closely together so as to resemble the tessellated paving of a street, on account of which they are spoken of as paving stones. These excrescences never invade the transitional folds, which remain normal in color, and they are seldom if ever to be seen on the conjunctiva of the lower lids, which is simply thickened. This is the typical picture of the **tarsal** variety of vernal catarrh, in which no changes are to be seen about the cornea.

In the **bulbar** form the excrescences on the tarsal conjunctiva may be few or absent, while yellowish gray, or reddish brown, semi-transparent nodules may be seen at the corneoscleral margin, where they overhang the cornea and slope away to blend with the bulbar conjunctiva. These may be found about the entire periphery of the cornea, or be confined to the region of the palpebral fissure. Their surfaces at a little distance from the cornea may appear dull and dry, and we may see near them some large, tortuous conjunctival vessels. In other cases the changes at the limbus consist of papillary elevations, a gelatinous appearing band, or a few red dots, but we are more likely to meet with these in the third, or **mixed** variety, in which the tarsal and bulbar forms are combined. In this mixed variety the typical changes on the lids and about the cornea may be present, or those in either location may preponderate over those in the other. Whatever the changes in the conjunctiva may be they persist almost without alteration summer and winter, whether symptoms are excited or not, and gradually undergo involution as the disease dies out. The most perfectly characteristic signs are the milky, opalescent discoloration of the conjunctiva, and the peculiar glutinous secretion, for the regular intermissions during cold weather may be omitted, or the symptoms may be at their height at this time.

Nothing is known concerning the cause of vernal catarrh. It is a sporadic disease, it is not contagious, and it occurs in all classes of people. Almost always it begins in childhood or youth, attacks males more than females, and lasts an indefinite number of years until it passes away spontaneously. It is met with in adults, and some writers claim that it may start even during middle life.

This disease is mistaken most often perhaps for trachoma, but it seems hardly possible for us to make such a mistake if we exercise reasonable care in making our observations, because *vernal catarrh leaves the transitional folds normal,* while these are the principal seat of the disease in trachoma, and the *bluish, milky tinge* of the conjunctiva peculiar to the former is not seen in the latter. It is possible for the two diseases to occur together in the same patient, but even then the flat paving stones of the tarsal variety of vernal catarrh do not look much like the granules of trachoma that lie embedded beneath the conjunctiva on the surface of the tarsus, while the marginal excrescences of the bulbar form differ altogether from any of the trachomatous affections of the cornea. There is a certain superficial resemblance between a phlyctenule and a pericorneal

nodule of vernal catarrh, yet the dissimilarity is apparent as soon as we compare the two conditions. A phlyctenule is accompanied ordinarily by other symptoms of phlyctenular conjunctivitis, in which the history is altogether different from that of vernal catarrh, while both the milky appearance of the conjunctiva, and the peculiar, stringy discharge are wanting, it develops rapidly, soon ulcerates, and is gone in a few days, while the pericorneal nodule is hard, of long duration, never breaks down, and is accompanied by the history and conjunctival appearances of vernal catarrh. The only cases in which differentiation is difficult are the rare ones in which the only prominent symptom is a single mass at the corneoscleral margin, when we may be obliged to excise it and subject it to a microscopical examination before we can decide whether it is sarcoma, an epithelioma, a tuberculoma, or an efflorescence of vernal catarrh.

TRACHOMA

Trachoma is a specific, contagious disease of the human conjunctiva characterized by slowly progressive changes that involve not only it, but the cornea and tarsus as well. The disease is scattered over the entire world, but is not distributed evenly; in some places it is endemic, in others it is rarely to be seen, while islets of varying size in which it abounds are to be found in regions that are free from the scourge. Wherever it is endemic it spreads slowly, infecting one person after another, yet not all. Prolonged daily contact seems to be necessary for the communication of the infection. If an individual suffering from this disease is shut up with healthy persons in an institution, some, but not all, of his associates will become infected in the course of time. If a man marries a wife who has trachoma he may become infected in less than two months, or not for several years, while part or all of his children may suffer likewise. The disease seems to be more contagious at some times than at others, but it is doubtful if it can be communicated during a brief sojourn with its victims, at least if such ordinary precautions are observed as to use exclusively one's own towels, handkerchiefs, bed linen, and wash basins. The specific agent has not yet been determined with certainty.

The **early stage** of the disease is marked by two characteristic features, a development of granules and a proliferation of the papillæ of the conjunctiva, both of which are present in every case,

though in varying proportions to each other. The papillæ may be so small that they can scarcely be detected without a microscope, or so exuberant as to mask the granules. The conjunctiva is hypertrophied as the result of an infiltration of its adenoid layer, which gradually invades the subtarsal tissue and the tarsus itself, and finally is transformed into cicatricial tissue. The disease may attack the corneal epithelium at almost any stage in its course. The clinical pictures differ widely, at first because of the proportionate preponderance of granules or papillæ, later according to the degree of involvement of the cornea and of the tarsus, or the extent to which cicatrization has taken place.

When a patient complains of a constant irritation of his eyes, that his lids are stuck together in the morning, or that he has a more or less profuse watery or mucopurulent secretion, while his upper lids droop a little, though presenting no signs of inflammation aside from a swelling that is most marked above, we think of a possible trachoma. In our routine inspection we may or may not find the semilunar fold red and swollen, but usually see a moderate injection of the bulbar conjunctiva, especially toward the transitional folds. If the photophobia is considerable we look for a reddish film over more or less of the upper part of the cornea. After the upper lid has been everted, which is not accomplished as easily as usual, the upper transitional fold protrudes and is seen to contain spawn-like **granules,** 1 to 2 mm. in diameter, arranged irregularly and extending nearly from one canthus to the other. Generally they are roundish in shape, grayish in color, opaque or semitranslucent, but sometimes they are confluent and form flat or fungoid masses. If we pinch this swollen fold between our fingers a gelatinous material escapes from some of the granules, which then become flattened. Scattered over the surface of the tarsus are many little, round, yellowish, or reddish yellow spots, which are similar granules that are unable to elevate the tightly adherent conjunctiva. Like changes are present in the lower transitional fold and in the palpebral conjunctiva of the lower lid, but the granules are much less developed, a point to be considered in the differentiation from follicular conjunctivitis. The disease usually starts either in the upper transitional fold, or in the conjunctiva of the upper lid, and always is best developed in that locality. *MacCallan* says that the palpebral conjunctiva is the point of attack in Egyptians, among whom the transitional fold often is spared, while it ordinarily settles in this

fold among Europeans. The granules may extend down into the bulbar conjunctiva, sometimes nearly to the cornea, and may involve the semilunar fold, in which the disease has been known to start. The conjunctiva usually is reddened, particularly about the bases of the granules, but sometimes it is pale; occasionally it is hard to say whether it is thickened or not, but probably it is in all cases. This is the form called **granular** trachoma, in which the enlargement of the papillæ is very slight.

Quite a different picture is presented when the papillæ proliferate exuberantly and produce that of **papillary** trachoma. In this the tarsal conjunctiva is strewn with fine, red elevations which are packed closely together and give it a moist, red, velvety appearance, or one that has been compared to that of sandpaper. These elevations are smallest near the margin of the lid and grow larger as we pass toward the upper edge of the tarsus, just beyond which they may be very large and produce growths that have been likened to cockscombs, cauliflowers, and red raspberries. These may hide the granules, which nevertheless are present and are to be found if looked for carefully. A similar hypertrophy of the papillæ follows gonorrheal ophthalmia, severe attacks of other forms of acute conjunctivitis, and appears sometimes in chronic conjunctivitis, especially when the latter has caused ectropion, but in the former the hypertrophy soon passes away and the history aids in the diagnosis, while in the latter they occur about the lower lid and no granules can be found. The last is an important diagnostic point, for in many cases of trachoma the hypertrophy of the conjunctiva forces the lower lid away from the eye and produces an ectropion associated with a much greater proliferation of papillæ than is to be found elsewhere.

Between these two extremes are many pictures of the so-called **mixed** trachoma, the commonest form, in which both granules and papillæ are visible, one or the other predominating more or less as a rule. As long as we can make out little yellowish spots in the tarsal conjunctiva, or there is any implication of the upper part of the corneal epithelium, the diagnosis is plain, but when we cannot be sure whether any such spots are hidden by the swollen papillæ or not, and when there is no affection of the cornea, we may have difficulty in deciding whether the case is one of trachoma, of follicular conjunctivitis, or of a development of follicles following an acute conjunctivitis. The problem is complicated a little by the

fact that it is possible for this disease to follow an acute inflammation of the conjunctiva and to present the picture known as acute trachoma. Formerly it was thought that trachoma was accustomed to set in with acute symptoms, but *MacCallan* states that this is rarely the case even in Egypt, which is perhaps the worst hotbed of trachoma and gonorrheal infections of the eye in the world, although it is admitted that a double infection of trachoma and acute conjunctivitis might be transmitted from a patient suffering from both. Such a double infection seems to be rare in the United States, and we do not expect trachoma to follow an acute conjunctivitis.

The eversion of the upper lid becomes increasingly more difficult as the disease progresses, because the upper part of the tarsus grows thickened and protuberant as the result of its infiltration. The granules are apt to show signs of confluence, and may coalesce into a gelatinous mass that gives a peculiar, hyaline appearance to the thick, firm, smooth, yellowish conjunctiva.

No matter which of these clinical varieties has been present in the early stages, lines of **cicatrization** appear sooner or later in the tarsal conjunctiva, cutting it up into islands of thickened tissue separated by narrow whitish bands. We may or may not be able to find granules at this time, but sometimes it seems as though a new formation of granules had appeared, associated with an exacerbation of the pannus, pain, photophobia, and lacrimation. The cicatricial bands grow wider, new ones appear, and the islands become smaller until they vanish, when the entire palpebral conjunctiva has been transformed into smooth, white cicatricial tissue, the wide upward sweep of the upper transitional fold has gone, so that the membrane can be seen to pass directly from the upper edge of the tarsus to the eyeball, and one or more symblepharons may be found between the lower lid and the globe.

Trachomatous Pannus

The epithelium of the cornea may become involved at nearly any stage of the disease, and pannus is certain to develop sooner or later. The first sign of such an involvement is a slight injection along the upper margin of the cornea, with a number of elevations which are so minute at first that they can scarcely be seen without the aid of a magnifying glass. A hazy film spreads downward from these in the corneal epithelium, and after this has progressed about

1 mm. little red vessels shoot down into it from above. As the process continues more and more of the upper half of the cornea becomes covered by a reddish gray cloud separated from the healthy tissue by a sharp, horizontal line of demarcation. *This pannus is characteristic and diagnostic of trachoma.* It may be differentiated from all other conditions by the facts that it starts from above, terminates below at a sharply defined horizontal line which crosses the cornea above, at, or just below its center, and that its vessels can be traced upward into the conjunctiva.

While it is fresh and its vessels few and small it appears thin, with a slightly wavy surface, and often we are able to detect places within it where the epithelium has been cast off, if we make a sufficiently close examination. This is called **pannus tenuis,** and may clear up completely. If the disease continues to advance, the redness is increased by the development of more new vessels, infiltrations and opacities form, the surface becomes uneven, and we have **pannus vasculosus,** recovery from which rarely is perfect. Still later in the disease, most often when the irritation produced by entropion and trichiasis has been added, the parenchyma of the cornea becomes involved, the pannus is distinctly raised, and looks like a layer of raw flesh, or of granulation tissue; this is **pannus crassus,** or **carnosus,** which undergoes cicatricial transformation instead of involution, after which it is known as **pannus siccus.** In very bad cases of pannus crassus some of the vessels, which spread over the entire cornea, may come from the sides or below, instead of all from above, as in the other varieties. Ulcers are quite apt to appear on the cornea at almost any time, usually at the margin of the pannus.

Sequelæ of Trachoma

The sequelæ of trachoma are impaired vision, entropion, trichiasis, and xerosis. The degree of each must be due to the extent to which the tissues involved have been destroyed, for if the destruction was complete in all cases the results would be the same, but they are not. When all of the glandular elements of the conjunctiva have been destroyed the patient suffers from the pitiable condition **xerosis,** in which the surface of the eyeball feels, looks, and is dry. Fortunately this is not common, even in patients whose palpebral conjunctivæ have been changed into smooth white cicatrices, and who can hardly be said to have any transitional folds; it is hard to say why, unless

we suppose that enough of the glands have been spared to keep the surface moist, for the dryness is not influenced by the lacrimal fluid, which may or may not continue to pass over it. In some cases the efferent ducts of the lacrimal gland are occluded by the cicatrization and the gland atrophies, in other cases this does not happen. The infiltration of the tarsus results in a softening of that tissue and an injury to the roots of the lashes, and then the cicatricial changes produce a distortion of the tarsus, yet the entropion and trichiasis differ in degree in individual cases. In one the lashes may have been reduced to a few stunted, colorless remnants, in another numerous normal cilia remain intermingled with those pathologically changed. About 3 mm. above the free margin of the lid, where the conjunctival cicatrix is densest, the tarsus is bent at an angle, which is more pronounced in one case than in another, and produces a correspondingly different degree of entropion in each, although the disease has seemed to run about the same course in both at the same time. The vision of one patient may be practically destroyed by a pannus siccus, while that of another may be useful in spite of scattered opacities and an irregular astigmatism of the cornea. A picture cannot be drawn that will present accurately the details in every case of the final stage, **cicatricial trachoma,** but when we meet with a patient who has a constant irritation of his eyes, which have been sore for years, whose palpebral fissures are shortened, whose upper lids are more or less deformed and very difficult to evert, who has more or less trichiasis, entropion, and an impairment of vision that is to be ascribed to irregular astigmatism, or to an opacity of the cornea, the cicatricial changes above described are pretty sure to be seen as soon as we have everted the lids.

BENIGN FOLLICULAR AFFECTIONS OF THE CONJUNCTIVA

When we evert the lower lids and find little roundish or oval granules or follicles, either few in number or densely packed, we are in no way justified in an immediate diagnosis of trachoma, for the conjunctiva reacts to a number of irritants of various kinds by the formation of such little growths. Yet if anyone maintains that any particular case of follicular trouble is one of trachoma, we shall have difficulty in demonstrating the contrary, because the only positive proof is to be obtained through the subsequent course of the disease,

and even this is not always accepted as proof by those who doubt the existence of more than one follicular disease. Simple folliculosis and follicular conjunctivitis, whether due to acute inflammation of the conjunctiva, drugs, or unknown causes, pass away completely and leave both the conjunctiva and cornea unharmed, while trachoma produces cicatricial changes. The differences in the clinical appearances are slight and merge into one another to such an extent that an expert often is in doubt as to the correct diagnosis, while the histological pictures are the same, and none of the ætiological agents to which trachoma has been attributed have been demonstrated to be present in every case, or to be peculiar to the disease.

Follicles Induced by Conjunctivitis

Sometimes follicles appear in the course of an acute catarrhal conjunctivitis, usually after the abatement of the inflammatory symptoms, whether it is of infective origin or not. *Greef* makes the point that they are not produced by gonorrheal or diphtheritic infections, or by a simple chronic catarrh, no matter how long it may last, and that their development seems to depend on the specificity of the stimuli, rather than on its intensity or duration.

We see a number of smooth, whitish red follicles from 1 to 3 mm. in diameter in the lower transitional fold, to which they are usually confined, though occasionally some are to be found in the upper, where generally they are smaller and fewer. When only a few are present they tend to gather about the canthi. Sometimes they are to be seen also in the semilunar fold. Swollen papillæ and other signs of a retrograding conjunctivitis are present. The only questions that arise are whether a folliculosis or a trachoma existed prior to the acute inflammation, and whether this may not be one of the rare cases of acute trachoma. If the case is one in which we know that no follicles or granules existed prior to, or during the early stages of the inflammation, the first question is answered promptly in the negative, otherwise we cannot be positive. If these formations are confined mainly to the upper transitional fold the disease probably is trachoma, because this is the favorite place for it to start, but if they are chiefly in the lower fold we may be pretty sure that it is not. Many cases are doubtful clinically, but follicles following acute inflammation are common, while acute trachoma is not.

Follicles Induced by Drugs

After we have treated an eye for some time with atropine, eserine, or an ointment of yellow or red oxide of mercury, a slight conjunctivitis frequently sets in, for which we can find no bacterial cause, accompanied by a development of follicles in the lower transitional fold. The conjunctiva over them usually is rosy in color, but not thickened. Sometimes the idiosyncrasy of the patient is so marked that a follicular catarrh is excited by a single application of the drug, but this does not occur often. Other drugs that have been known to produce the same effect are cocaine, homatropine, duboiscine, hyoscyamine, and arecoline. These cases prove that the development of follicles may be excited by chemical as well as bacteriological stimuli.

School Folliculosis

When we evert the lower lids of a school child and find the lower transitional folds full of little rounded follicles, with no signs of inflammation, we must not jump to the conclusion that he has trachoma, for the chances are that he has not. Yet this mistake is common, and thousands of eyes have been operated on unnecessarily. First, we must make sure whether the upper transitional fold is implicated more or less than the lower, whether granules can be seen on the tarsal surface of the palpebral conjunctiva, and whether there are any signs of irritation of the cornea. If the upper transitional fold is full of large pale granules which have spread into the tarsal conjunctiva, the disease probably is trachoma, and this diagnosis is made absolutely certain by the presence of some minute blood vessels extending into the epithelium from the upper margin of the cornea; but *if the follicles are small, congregated mainly in the lower transitional fold, do not encroach on the tarsal surface of the conjunctiva, the cornea is unaffected, and there is no conjunctivitis, the child has simple or school folliculosis.*

Wherever children spend hours daily crowded together, as in a schoolroom, a certain number will be found to have little round or oval translucent follicles, sometimes yellowish in color, in the lower transitional folds, that arrange themselves in rows when the lower lids are everted, with fewer and smaller ones in the upper folds. They are apt to be rather crowded near the canthi, but each follicle is separate from the others and there is no tendency for them to

blend. Both eyes are affected, but are absolutely not irritated. The conjunctiva over the follicles is normal in appearance, or perhaps shows a slight redness that extends a little way into the bulbar conjunctiva, but we cannot say that there is any conjunctivitis. Such a folliculosis is met with in private schools attended only by the children of the wealthy, but not so commonly as in the more crowded public schools. It is found in the city and the country, in every grade of society. *Ray* claims that it is present in from ten to twenty-five per cent. of all children in institutions. *Stephenson* found it in the great majority of nearly 15,000 school children, and believes that a follicular condition is natural to the conjunctivæ of many young persons. We seldom see it except in children, but *Greef* maintains that it develops wherever many persons are crowded together, and that it can be detected with the microscope in the conjunctivæ of all prisoners who have been confined for a long time.

The cause is uncertain. Many believe it to be an expression of adenoid activity incident to childhood, as it often is associated with enlarged tonsils and adenoids and undergoes involution at the same time with these. The suggestion has been made that many cases of tonsils and adenoids are the result of faulty secretions of the endocrinic glands, and if this is true the same cause may operate here. *Greef* ascribes it to an irritant contained in the stagnant air of a crowded place, perhaps ammonia, perhaps an anthropotoxin. It is not regarded generally as communicable, though *Axenfeld* did succeed in exciting a transient follicular trouble in his own eye by inoculation from an endemic either of this or of follicular conjunctivitis in an orphan asylum. Whatever the cause may be, school folliculosis gets well spontaneously after an indeterminate length of time, usually before the children attain their growth, and leaves no traces. *Greef* says that it frequently disappears during the summer vacation, but such an evanescent case has never come under my observation.

Follicular Conjunctivitis

When a reddened, somewhat thickened conjunctiva, with enough discharge to at least glue the lids together in the morning, is associated with follicles in a child, it may be difficult to decide whether the case is one of follicular conjunctivitis or of trachoma. Much difficulty is experienced in studying the literature of follicular troubles of the conjunctiva because most writers use the terms folli-

cular conjunctivitis and folliculosis interchangeably, even though they may speak of inflammatory and noninflammatory types, and often it is hard to tell of which type they are speaking. We should avail ourselves of every point in the differentiation of these troubles which resemble trachoma so closely, and it seems as though a form which runs its course without irritation of the eye, or any essential change in the conjunctiva, presents sufficient clinical characteristics to warrant its separate consideration as a folliculosis. But it is only fair to say that many competent observers question whether both folliculosis and follicular conjunctivitis are not manifestations of one and the same disease, trachoma, in spite of the fact that a distinction has long been recognized. A necessary corollary of this belief is that the disease is one in which recovery takes place very often during the early stage with no permanent harm to the tissues, which places us in quite as bad a dilemma, how to distinguish between the benign and the malignant forms of trachoma before harm has been done.

We need to take into account whether trachoma is **endemic** in the particular locality in which the patient lives; this is not a sure sign, as sporadic cases occur of both diseases, but it is of assistance. If the follicles are confined mostly to the lower transitional folds, are grouped toward the canthi, show no tendency to coalesce, are absent from the palpebral conjunctiva, and the cornea is unirritated, we may feel pretty sure that the disease is follicular conjunctivitis, particularly if trachoma is not endemic in that locality. In many cases however the follicles abound in both cul-de-sacs, their size is not a reliable guide, we cannot be sure that some of them do not show a tendency to become confluent, or that the thickening of the conjunctiva is not due to a trachomatous infiltration and development of papillæ which conceals minute granules on the tarsal surface, and then we are in doubt as to the diagnosis. The differentiation in such cases can be made only from the course; if recovery takes place under conservative treatment, with no damage to any tissues, we call the disease follicular conjunctivitis; if cicatricial changes, or corneal complications appear, we pronounce it trachoma. We should determine if we can whether the case is one in which a formation of follicles followed an acute conjunctivitis, or one in which a catarrh was grafted on a folliculosis, a task that may be easy or difficult, but usually is of much less importance than the differentiation from trachoma.

TUBERCULOSIS OF THE CONJUNCTIVA

When a patient presents an œdema of the upper lid, or of both lids, of one eye, with a tender, swollen preauricular gland, perhaps enlarged and tender retromaxillary, submaxillary, parotid, and cervical glands as well, and states that some two or three weeks back the eye became red with a moderate amount of secretion, the lids began to swell nearly synchronously with the appearance of a lump in front of the ear, but with little or no pain, we suspect at once the presence of one of two diseases, Parinaud's conjunctivitis or tuberculosis. The descriptions of these two diseases often are confounded, for some still believe the former to be an atypical tuberculosis of the bovine type, and while there seems to be good reason to doubt this theory, we cannot deny that it is possible for tuberculosis to assume the same clinical form.

If we find on eversion of the lids that the entire conjunctiva is red and swollen except at one place where there is an irregular, dirty yellowish ulcer with raised margins, with perhaps some yellowish gray nodules in its vicinity, we may feel pretty sure that we have to deal with tuberculosis. Such an ulcer usually is found on the palpebral conjunctiva, *Fuchs* says generally in the subtarsal sulcus of the upper lid, but it seems to occur nearly as often in the lower fornix, and may appear on the bulbar conjunctiva. It may start as a number of miliary ulcers that coalesce, or as a rough thickening which breaks down, and is apt to spread superficially, though it may remain stationary, or heal spontaneously in exceptional cases. Sometimes it penetrates deeply into the tarsus, or even through the lid, or into the eyeball.

An ulcer like this usually is found associated with lupus of the face, or tuberculosis of the adjacent tissues, the nose, larynx, or some other portion of the body, but sometimes it is primary. The **differentiation** from most of the other ulcers of the conjunctiva is not difficult. A syphilitic ulcer is rare, and the enlarged preauricular gland is hard and not tender. An ulcer caused by traumatism or a burn might be associated with an enlarged, tender gland, and so might one left by a membranous conjunctivitis, but a history of injury, or of a violent inflammation would mark such a case. The ulcers of pemphigus are gray, cicatrize, and new bullæ appear, while the disease is present in the face, as a rule, and it would be excep-

tional for the lymphatic glands to be swollen. The open sore left by a ruptured chalazion can be differentiated by the hard mass to be felt over it in the tarsus. There remains only the form of Parinaud's disease which is characterized by ulcers of the conjunctiva, and a tuberculin test may be necessary to exclude this.

The clinical pictures produced by tuberculosis of the conjunctiva vary with the susceptibility of the patient, the reaction of the tissue, and the virulence and numbers of the bacilli, just as elsewhere in the body, so instead of a plainly visible ulcer we may see only a florid, diffusely swollen conjunctiva that contains many little, grayish, miliary nodules beneath its epithelium; or red, hypertrophied papillæ that cover the palpebral conjunctiva and spring from the transitional folds; or grayish yellow nodules which resemble trachoma granules, mixed perhaps with enlarged papillæ; or one or more red, pedunculated excrescences, usually coming from one or other transitional fold. All of these resemble Parinaud's conjunctivitis, and some of them trachoma. The probability of the latter becomes very slight when we remember that trachoma rarely affects one eye alone, and that it is still more rarely associated with swellings of the lymphatic glands, though it may be at least theoretically possible for a trachoma of one eye to coexist with glandular tuberculosis of the same side. In some of these cases the differentiation can be made from the lesions on the cornea. Occasionally a sort of pannus develops, but instead of presenting a reddish film which extends downward over the upper part and is delimited below by a horizontal line, it enters the cornea from any direction and presents nodular elevations, granulations, or ulcerations. The presence of such a pannus is a strong indication of tuberculosis and against either trachoma or Parinaud's conjunctivitis.

A portion of the tissue should be excised in every case, part to be examined microscopically and bacteriologically, part to be injected into the abdomen of a guinea pig to see if it can give rise to tuberculosis. A tuberculin test should be made. In young children a von Pirquet's test may be satisfactory, especially if it is negative, but as positive reactions are obtained almost invariably in older children, and in adults, it is better to rely on the subcutaneous injection. If the presence of tuberculosis is demonstrated by these means the diagnosis is made.

PARINAUD'S CONJUNCTIVITIS

This rare disease sets in with a reddened eye, usually some secretion, more or less fever, and a rather slowly developing swelling of the upper lid and of the preauricular gland on the same side, so that at the end of about two weeks the patient presents ptosis, a large, more or less tender swelling in front of the ear, swollen cervical glands that grow smaller downward, enlarged and tender retromaxillary, submaxillary, and perhaps parotid glands, all of which contribute to give a swollen appearance to the side of the face and neck, much like that produced by tuberculous glands. The bulbar conjunctiva is reddened, perhaps chemotic, and when we evert the lids we find proliferations on the conjunctiva that vary in different cases. Sometimes we see large, reddish or yellowish, semitransparent, polypoid growths, which may be pedunculated; sometimes large or small, red or yellowish granules; sometimes both of these intermingled; sometimes red, buttonlike elevations surmounted by yellow points; sometimes a fluffy and uneven conjunctiva covered with little white spots, and sometimes ulcers of the conjunctiva with granulations in their vicinity. These changes may be scattered over the entire conjunctiva, including the caruncle and the semilunar fold, but more commonly are confined to a certain portion and exhibit a preference for the upper transitional fold. In one case of my own the upper fornix was occupied by a mass of acuminate growths with yellow points, over which the conjunctiva passed smoothly except at the apex of one, where it was ulcerated. No pus could be obtained from the yellow tissue. In another there was a little gray ulcer in the outer part of the bulbar conjunctiva which was almost concealed by large, pointed granulations in the immediate vicinity, which were most pronounced in the outer part of the lower transitional fold.

One eye alone is affected almost invariably. The conjunctival lesions reach their acme and then slowly undergo involution. The swollen glands may likewise undergo involution, or they may break down and suppurate. The conjunctival symptoms pass away in a few weeks, but the swelling of the glands, when no suppuration occurs, may persist for from two to six months.

When large yellow granules, or polypoid growths that resemble enlarged papillæ, or both of these combined, occupy the upper transitional fold and the palpebral conjunctiva, it may not be easy to

exclude a unilateral trachoma with a coincidental enlargement of the lymphatic glands on the same side, but such a combination is scarcely more than theoretically possible. The diagnosis of trachoma should not be made unless it is supported by the presence of a trachomatous pannus, or by the histological examination of a piece of the excised tissue. The differentiation from conjunctival tuberculosis is more difficult. The histological characteristics are not quite the same in the two diseases, but to make a positive diagnosis sometimes we have to rely on exclusion of the latter by microscopical and bacteriological examination, by the tuberculin test, or by the subsequent course of the disease, for Parinaud's conjunctivitis will recover whether treated or not, and we do not expect this result in tuberculosis of the conjunctiva. Recently *Verhoeff* has found a hitherto unknown, minute, filamentous microorganism present in great abundance in the areas of cell necrosis in eleven out of twelve consecutive cases. In one early case numerous masses were found also in the superficial lymph spaces, especially just beneath the epithelium, where areas of cell necrosis usually occur. It is quite possible that he has discovered the active agent of this disease, and the means for making a positive diagnosis in the future. We have yet to learn how this microorganism obtains lodgment in the human body.

The disease is sporadic, met with at all ages, and the cause is not determined. It has been ascribed to animal origin, to infection with foot and mouth disease, and is believed by some to be a manifestation of bovine tuberculosis, but it occurs in persons who give no history of animal contact, in regions where foot and mouth disease has been unknown for many years, and the presence of tuberculosis in any form has not been demonstrated satisfactorily. A very few cases have been reported in which some yellowish gray granules in the palpebral conjunctiva of the upper lid proved on microscopical examination to be masses of actinomycosis, or sporotrichosis.

PHLYCTENULAR CONJUNCTIVITIS

When we see one or more roundish, or oval, whitish gray nodules in the conjunctiva at the margin of the cornea, each at the apex of a triangle of hyperæmic vessels, which attains its growth in a few days, then breaks down into an ulcer that gets well very quickly, we call each nodule a phlyctenule. The rest of the conjunctiva

may be perfectly normal, but the number, size, and situation of the phlyctenules vary a good deal and give rise to varying pictures. The number may range from one to a great many, each with its own triangle of injection, so that the eyeball may be red only in spots, or present a generally reddened appearance. The more numerous they are the smaller they become, so when we see the cornea surrounded by a band of bright red conjunctival vessels, with no pain or secretion, we will be apt to find a granular appearance about the limbus due to a multitude of phlyctenules so minute that they can scarcely be seen separately. Occasionally phlyctenules appear at a distance from the limbus and then are centers of zones of injection. Rarely a single one attains a large size and may cause us some worry, for it persists longer than smaller ones and is accompanied by much more hyperæmia; our minds are relieved when its surface ulcerates and it passes away. A phlyctenule at a distance from the cornea needs to be differentiated from an episcleritis, sometimes from a nævus, or a small ecchymosis. An **episcleritis** is associated with pain and the injection is of the episcleral vessels, while a phlyctenule is without pain and the injection can be seen to move with the conjunctiva; the former is apt to persist for a long time, the latter soon departs. A **nævus** has existed long and consists of a little congeries of vessels, while the phlyctenule appears suddenly and lasts but a short time. No vessels are visible in an **ecchymosis.**

Most cases of phlyctenular conjunctivitis are associated with keratitis, so the claim that the disease is a keratoconjunctivitis seems to be justified. The serious lesions are produced in the cornea and will be discussed under **phlyctenular keratitis,** but the disease should not be considered a trivial affection, even when it is confined to the conjunctiva, for it tends to recur and to involve the cornea sooner or later. It occurs mostly in children, sometimes in adults, but almost always in those who have had attacks during childhood.

HERPES SIMPLEX

Rarely we see clusters of vesicles appear on the bulbar conjunctiva and rupture quickly, leaving shreds of epithelium hanging about the margins of small ulcers that heal in a few days. They are associated usually with a simple herpes of the lids, or of the lips, and ordinarily appear in the course of digestive or respiratory disturbances. The cornea may be involved with the production of photo-

phobia and lacrimation. This condition is differentiated from phlyctenular conjunctivitis by the absence of phlyctenules and the presence of shreds of epithelium; from herpes zoster by the absence of severe trigeminal neuralgia and the characteristic eruption on the skin; from pemphigus by the absence of this disease on the face, of successive crops of characteristic bullæ, and of cicatrization. It is thought by some that the presence of simple herpes indicates trouble in the ciliary ganglion, but this is not yet established.

PEMPHIGUS

In this extremely rare disease the patient complains of severe pain in his eyes, photophobia and lacrimation, and we find gray patches or areas of cicatrization, or else bullæ that rupture quickly and leave gray ulcers. The bullæ appear for a long time in successive crops, while the ulcers cicatrize slowly, replacing the conjunctiva involved with cicatricial tissue which shrinks. The disease is almost always bilateral and to be found on the lids, or elsewhere on the body. Its course cannot be checked, and it results in a total cicatrization of the conjunctiva, that produces entropion, trichiasis, and symblepharon. When the last is total the xerotic, cicatricial conjunctiva may be seen to pass directly from the margins of the narrow palpebral fissure to the anterior surface of the eyeball, and there may be ankyloblepharon, especially at the inner canthus, where the disease is apt to begin. The cornea seldom is spared, but is likely to become ulcerated and staphylomatous, or to have its surface covered with a growth that resembles pannus. Finally xerophthalmos develops.

SYPHILITIC LESIONS OF THE CONJUNCTIVA

Chancres are rare, but occur most often in the lower transitional fold and on the conjunctiva of the lower lid. The patient first notices a growing, painless swelling of the lid, and when the latter is everted we see a dirty whitish red tumor, perhaps as large as a bean, with an ulcerated surface covered with fibrin. The rest of the palpebral conjunctiva is red, and the neighboring bulbar portion may be slightly chemotic. The tumor is felt through the lid to be very hard, and the preauricular, or the submaxillary lymphatic gland is swollen, hard, and painless.

The lesions for which a chancre may be mistaken are an epithelioma, a tuberculous ulcer, a gumma, or a chalazion, if it is in the right situation, and a positive diagnosis can be made only through the finding of spirochætæ in scrapings from its surface, or by waiting for the secondary symptoms of syphilis to appear. The latter course is hardly justifiable, unless the specific microorganisms have not been found after a careful examination by an expert, and we are not allowed to excise a portion for examination, because the probable diagnosis is not difficult as a rule. A chancre usually is met with in young persons; an epithelioma in the elderly, generally at the corneoscleral margin, grows more slowly, and the lymphatic glands are not likely to be affected. A tuberculous ulcer is not apt to have a hard base; unless it has been much irritated by treatment, and the preauricular gland is softer and tender. A chalazion is attached immovably to the tarsus, while a chancre is not, and it seldom if ever causes an enlargement of the lymphatic glands. A gumma is much more rare, and is likely to be associated with a positive Wassermann. The Wassermann test cannot be positive in the case of a chancre until the disease has become diffused through the system, when the secondary symptoms appear.

Mucous patches like those seen in the mouth occur on the conjunctiva during the secondary stage of syphilis, or papules may appear on the bulbar portion, the caruncle, or the semilunar fold, and leave brownish spots when they heal.

The descriptions given of a **gumma** of the conjunctiva do not quite agree. *Ball* says that they are rapidly growing, rounded, smooth, firm tumors of a light pink color and the size of a split pea, which cause no pain when not complicated, and quotes *De Beck* as saying that they "usually are developed in the ridge where the conjunctiva passes into the cornea, but may occur in other parts of the membrane." *Roemer* says that their general color is a dull red with yellowish gray on the top and margins, and that they excite much irritation of the eye, into which they extend deeply, while serious ulcerations are apt to take place. Probably both authors are right as regards individual cases, but it is doubtful if a diagnosis can be made with certainty except by the observation of the effect of treatment, or by an examination of excised tissue. A Wassermann test should be positive, but this would not exclude the development of some other form of tumor in a syphilitic, and a gumma would not be excluded absolutely by a negative result.

AMYLOID AND HYALINE DEGENERATIONS OF THE CONJUNCTIVA

These conditions are very rare in the United States. The lids gradually grow thick and heavy until the patient can no longer open his eyes and he presents extreme ptosis with no external signs of inflammation. The lids are stiff, hard to separate, still more difficult to evert. The bulbar conjunctiva may appear to be normal or wax-like; the cornea usually is clear. On eversion of the lids we find that their entire conjunctivæ have been changed into a swollen, smooth, friable, waxy mass, which bleeds very little when torn, with some elevations perhaps toward the transitional folds.

We are unable to tell whether this is an amyloid or a hyaline degeneration of the conjunctiva except by excising a piece of the tissue and ascertaining its chemical reaction, for the two conditions resemble each other in every other respect. Such a degeneration is met with only in adults, and may be unilateral or bilateral. Some of the patients have had trachoma, and in these the cornea may have suffered, but there seems to be no ætiological connection between these degenerations and this disease. It is said that the process may go on to calcification or ossification of the conjunctiva.

CALCIFICATION OF THE CONJUNCTIVA

Leber described in 1893 an extremely rare disease or degeneration that he called conjunctivitis petrificans. During an attack of conjunctivitis white spots appear in the palpebral conjunctiva, and the epithelium over the larger ones is cast off, laying bare hard masses of lime, probably in organic combination with a base consisting of degenerated connective tissue. More attacks follow and more lime is deposited during a period of months or years, until a large amount is present. *Saemisch* removed 2 ccm. from one patient who had suffered three attacks. The cause is unknown.

While the deposit of such large quantities of lime in the conjunctiva is one of the greatest of rarities, that of small quantities is common. We see little yellowish deposits scattered irregularly over the palpebral conjunctiva, or in the folds, each the center of a little spot of hyperæmia, where they may be either the cause or the result of a chronic inflammation, but much more often they are to be

found in the acini of the Meibomian glands, where usually they are symptomatic of the uric acid diathesis; this is commonly called lithiasis of the conjunctiva. The patients complain of a scratchy feeling, like that produced by a foreign body, and are subject to a chronic conjunctivitis which cannot be cured until these small masses of lime, uric acid, or sodium urate, have been removed.

LEPROSY OF THE CONJUNCTIVA

One or more nodules on the conjunctiva associated with an abnormal whiteness or loss of the eyebrows or lashes, infiltrated or nodular lids, and perhaps similar nodules in the cornea and iris, are quite suggestive of leprosy. The diagnosis is made by finding leprosy bacilli in a nodule, or in the juice obtained from an incision into an infiltrated place in the lid. An interstitial keratitis with iritis is said to be not uncommon, and sometimes multiple, rarely single, nodules appear in the iris and partially fill the anterior chamber.

BURNS OF THE CONJUNCTIVA

The diagnosis of a severe burn of the eye is easy. The history of an accidental introduction of lime or other caustic, molten metal, hot fat, hot water, or steam, into the eye usually is quite definite; the swollen lids and the chemosis testify to its severity. We may find lime or metal present, or see grayish white patches where the conjunctiva has been burned to form eschars, which later are cast off so as to leave raw surfaces which cicatrize and agglutinate to form symblepharons.

More superficial burns produced by milder caustics, like carbolic acid, acrid vapors or liquids, and the flash of electric light from a short circuit, or electric welding, or by prolonged exposure to a powerful electric light, excite an acute conjunctivitis of short duration, but accompanied by severe pain, photophobia, lacrimation, reddening of the conjunctiva, sometimes chemosis and œdema of the lids. An exactly similar picture is seen in snow blindness, which is caused by the reflection of the sun's rays from snow that induces a loss of the corneal epithelium.

All degrees between these two extremes are met with. In the first certain areas of conjunctiva are permanently destroyed, in the other the injury seems to be limited to the epithelium, just as in

burns of the skin. The prognosis in burns with acids and alkalies generally is in accordance with the nature of the agent, yet quick attention may be remarkably efficient in limiting the effects even of the worst, while mild ones may produce untoward results. Quick lime is extremely destructive, yet immediate proper treatment may cause it to result in nothing worse than pain and inconvenience for a few days. Carbolic acid generally effects only a trifling and temporary injury, yet I have known an eye to be permanently blinded after a bath of it, and at the same time have seen nothing worse than a very superficial burn produced by nitric acid, and by a solution of caustic potash, in cases in which proper treatment had been applied immediately.

CHAPTER VIII

The Cornea

The normal cornea is smooth, lustrous, very sensitive, and perfectly transparent everywhere, except at the limbus, where it joins the sclera. It is the first tissue of the dioptric system through which light enters the eye, and is therefore not only the site of visible lesions, but is also productive of visual symptoms whenever its transparency is impaired, or its surface roughened, and of visual symptoms alone when its curvature varies from the normal. Although we recognize the fact at once when a cornea is congenitally too small or too large, its normal size varies so much in different individuals that exact measurements cannot be given. According to *Priestley Smith* the average horizontal diameter at the base is 11.6 mm., but it must be remembered that this is an average. The vertical diameter at the base seldom is the same as the horizontal. When these diameters are the same the radii of curvature are equal and light passing through is focussed toward a focal point, but when they are unequal the light is focussed toward two linear foci, one in front of and at right angles to the other; this is regular corneal astigmatism. When the vertical diameter at the base is less than the horizontal, the vertical linear focus is in front, and we say that the astigmatism is with the rule; when the vertical diameter is the greater, the horizontal linear focus is in front and the astigmatism is against the rule. The radius of curvature usually is between 7 and 8 mm. A long radius is one of the factors in the production of myopia, a short one in that of hypermetropia, as these bring the focus nearer to, or farther from the cornea. These conditions may impair the vision, but can be corrected by glasses. Other conditions of the cornea that affect the vision, but cannot be corrected satisfactorily in this manner, have to do with its form, surface, and transparency.

IRREGULAR ASTIGMATISM

When the play of light and shade seen in retinoscopy is more or less confusing, especially near the point of reversal, and the reflec-

tion of the mires of the ophthalmometer are distorted, broken, or shattered, we shall find an irregularity of the surface of the cornea that reflects and refracts light in all directions. This irregular astigmatism presents no uniformity, is almost if not quite always of pathological origin, and cannot be corrected satisfactorily by glasses, because the refraction varies in different parts of one and the same meridian. The **impairment of the vision** depends on the degree to which the surface of the cornea has been roughened, and the situation of the unevenness, for a slight irregularity near the margin may exert no appreciable influence, while a similar one at the center may disqualify the eye for many purposes. An uneven surface often is associated with cicatricial opacities, and the combination may rob the eye of useful vision. We can detect irregular astigmatism with certainty by observing the reflection from the surface of the cornea of some regular object, like a window frame, or the black and white circles of Placido's disk; when the surface is smooth the reflection of such an object appears to be perfect in form, though much reduced in size, just as in a reflection from a convex mirror, but when the surface is not smooth it is distorted.

KERATOCONUS

Sometimes we get a confused picture with the retinoscope in which the center of the pupil is myopic, but the myopia lessens rapidly toward the periphery and may pass over into hypermetropia. This can be produced by only three conditions,—a senile change in the lens that occasionally precedes the development of cataract called false lenticonus, true lenticonus, and a conical cornea or keratoconus. If we then use the ophthalmometer and find a peculiar distortion of the mires in which they appear to be small in the center and to enlarge in every direction, we have located the trouble in the cornea, and when we view this tissue in profile we shall see that it is conical. This abnormal curvature is perceived readily when the condition is well marked, but when it is slight our attention may need to be drawn to it in this way by the peculiar refractive error, or the distorted reflection from its surface.

Keratoconus may be congenital, when it is apt to be associated with other developmental faults, but generally it appears about the age of puberty, principally in women. Its cause is obscure. It may be present in one or both eyes. The center of the cornea is thin,

has yielded to the intraocular pressure, and when it is very thin we may be able to detect a rhythmic, pulsating movement at this point. An opacity at the apex of the cone is not uncommon in well marked cases.

KERATOGLOBUS

A few cases have been reported in which congenitally globular corneæ were met with in eyes that were otherwise healthy and capable of good vision, but such cases are very rare. Almost always a globular cornea is a symptom of the abnormal enlargement of the eyeball known as hydrophthalmos, buphthalmos, or infantile glaucoma. All other protrusions or enlargements of the cornea are of pathological origin and will be considered under staphyloma.

Pathological conditions decrease the transparency of the cornea by the formation of more or less dense opacities, either spread diffusely through the tissue, or confined to circumscribed areas, and may affect its sensibility.

ANÆSTHESIA OF THE CORNEA

While it is possible that the exquisite sensitiveness of the cornea may be increased pathologically, such a condition of **hyperæsthesia** is very difficult to detect, and is of no known symptomatic value.

A total or a partial loss of the sensitiveness of the cornea on the contrary is an important diagnostic symptom, as it indicates a paralysis or paresis of the trigeminus, a numbing of its terminal filaments, or general narcosis. We test the sensitiveness by observing the lid reflex on touching the cornea with some object, like a bit of cotton, or the tip of the finger, or having the patient compare the sensation with that felt when the other cornea is touched in the same way. We must always be careful not to injure the epithelium, for a slight abrasion may give rise to serious trouble. When the sensitiveness of the cornea is normal the least touch excites a powerful reflex closure of the lids; if this reflex is delayed or weak the sensitiveness is reduced, if it is absent the anæsthesia is complete. This reflex is utilized frequently to determine how deeply a patient may be under the influence of ether or chloroform. Another sign that is present when the corneæ of both eyes are anæsthetic is that winking becomes abnormally slow, and sometimes is so nearly abolished that the epithelium is allowed to become dry; this constitutes

a serious danger when a local anæsthetic, like cocaine or holocaine, is instilled into both conjunctival sacs simultaneously, unless measures be taken to see that the eyes are kept moist.

If the patient is conscious and no local anæsthetic has been instilled, anæsthesia of the cornea usually is a ***sign of serious trouble*** that needs an immediate diagnosis. Sometimes we meet with it in hysteria, when it is associated generally with hemianæsthesia, amblyopia, amaurosis, or other hysterical symptoms, but in these cases it is not apt to induce ulcerative lesions. If the cornea is hazy as well as anæsthetic and the tension of the eye is increased, we are dealing with a case of glaucoma. If the patient has been suffering from intense facial neuralgia with lacrimation and photophobia of the eyes, we expect to find at least some traces of the characteristic eruption of herpes zoster on the face. Leprous spots on the lids may lead to a diagnosis of leprosy. When a reduced sensibility in both eyes is associated with a feeling of constriction of the muscles of the face we should investigate the central nervous system to ascertain whether this may not be Charcot's tabetic mask, a symptom of tabes. When the cornea presents a central ulcer, or a central cloudy area not yet denuded of epithelium, with a circumcorneal injection, and yet the patient does not suffer from pain, photophobia, or lacrimation, it is pretty sure to be anæsthetic, and then we make a diagnosis of neuroparalytic keratitis.

PIGMENTATION OF THE CORNEA

All opacities in the cornea appear to be black when seen in the light reflected from the fundus with an ophthalmoscope, but most of them are white or gray when viewed by oblique illumination. A little spot that is black when seen in this way, or with ordinary light, suggests a foreign body, even when the usual irritation of the eye is absent, although a puncture stained with India ink is a bare possibility, but we must be sure that we are not looking at a dot of pigment in the iris. If we should happen to see near the center of each cornea a small brown elliptical spot, with its long axis vertical, we should remember that a few cases of such congenital pigmentation have been reported. Examination with a magnifying glass will resolve the spot into a multitude of minute dots.

A **rusty brown patch,** irregular in shape and size, occasionally is seen after a hemorrhage into the anterior chamber, the coloring

matter from which has permeated the cornea, or more rarely after a hemorrhage into the cornea itself. Such a patch is apt to be central, or in the lower part, less often above, with a clear periphery as a rule, though it may cover the entire tissue. Absorption is most active in the periphery near the vessels, and proceeds slowly until after months or years the stain may disappear. The conditions from which such a discoloration needs to be differentiated are an anterior chamber filled with blood, a total dislocation of a black cataract into the anterior chamber, and siderosis. When there is no clear corneal tissue it may not be possible to tell whether we are looking at blood or a blood stain, or both combined, except by reference to the history, but this can be determined as soon as a portion of the cornea becomes clear. A black cataract dislocated into the anterior chamber, so that it lies evenly in contact with the posterior surface of the cornea, produces a picture exactly like that formed by a central stain of the latter with a regular margin and surrounded by a clear zone, through which little or nothing of the anterior chamber or iris can be made out. When a cataract is tilted so as to allow us to perceive an oval pupil, a tremulous iris, and an inequality in the depth of the anterior chamber, the diagnosis is not difficult, but when none of these things can be seen, it may be hard to make if the patient is elderly. If the eye was known to have had good vision prior to the accident which occasioned either a hemorrhage or a dislocation, the fact weighs heavily in favor of the former. The margin of a blood stain is less apt to be regular than that of a lens. Possibly we may obtain with the ophthalmoscope a light reflex by which the dark edge of a lens may be made out, or we may be able to see a layer of clear tissue apparently as thick as the normal cornea in front of the opacity, but the really diagnostic symptom is the presence or absence of increased tension, for a lens dislocated into the anterior chamber excites secondary glaucoma within a short time, while a blood stain is not likely to be associated with such a condition. The only other thing to be excluded is the very rare siderosis of the cornea induced by a particle of iron or steel within the eye. *Ball* observed a case of this nature in which opaque spots of a dark brown color were formed in the cornea as the result of the lodgment of a piece of steel in the lens. The history of such a case, the absence of blood in the anterior chamber or cornea prior to the appearance of the stain, the tendency of the latter to increase, and perhaps the detec-

tion of an intraocular foreign body, suffice to mark its character. Sometimes we see a yellow stain in the cornea of an atrophic eye which is due to degeneration.

OPACITIES OF THE CORNEA

Nearly every lesion of the cornea constitutes an opacity, as it becomes visible by rendering the tissue less transparent; the principal exceptions are an abrasion of the epithelium that can be seen only by light reflected from its margins, and an indolent ulcer, the base and walls of which are transparent. We differentiate the nature of an opacity through its relation to the surface of the cornea and the presence or absence of signs of irritation of the eye. We estimate its depth by throwing light upon it and viewing it from the side, when we can see whether it is on the surface, or is separated from this by a thin or thick layer of clear tissue. If the layer is thin the opacity evidently is in the tissue of the cornea, and we say it is deep, interstitial, or parenchymatous; if the layer is thick, the opacity is very deep, in the region of Descemet's membrane, but we cannot always be certain whether it is on the posterior surface or not. An opacity may be large or small, even punctate, diffuse or circumscribed, dense or so faint as to be scarcely visible, and may or may not be vascular.

A slender red line in or on the cornea is a newly formed vessel, which always indicates a pathological condition, for the cornea contains no vessels normally. A single one is seldom seen, usually many are present, and they vary much in number and size. They may be superficial or deep, and occur in all forms of keratitis, though often they are so small as to be visible only with the aid of a bright light and a magnifying glass. The opposite of this extreme is seen when they cover the entire cornea with a blood red mass, or form a salmon patch in its parenchyma, but the vascularity is not so great as this in the majority of cases.

When we see a grayish or white, superficial opacity in which there is no loss of substance, accompanied by a ciliary injection, we know it to be an **infiltrate** if it is not vascular, or some form of **pannus** if little red vessels abound in it. If such an opacity is without a ciliary injection and is not vascular, it is either a **cicatrix** or the result of **degenerative changes;** if it is vascular, it is an old pannus. If the spot shows a loss of substance, we

recognize an **ulcer;** if the cornea is surrounded by a ciliary injection, the ulcer is active, whether it is vascular or not; but if there is no ciliary injection, it is indolent, or in the process of healing. An opacity in the parenchyma associated with a ciliary injection indicates an **interstitial inflammation,** whether it is vascular or not; if it is circumscribed, yellowish, and attended by violent inflammatory symptoms, it is an abscess of the cornea, or onyx. A parenchymatous opacity without a ciliary injection has been left by a past interstitial inflammation. Punctate spots on the posterior surface of the cornea with a ciliary injection call attention to a **cyclitis.** A spot on a baby's cornea that may vary from a faint cloud to a dense leucoma, but is not associated with a conjunctivitis or an infantile glaucoma, is an opacity that probably has been caused by an **intrauterine inflammation,** though possibly it may be due to a fault of development. If there is any ciliary injection we know that inflammation still is present, but in any case we should try to determine whether it is superficial or deep, for if it is in the parenchyma it indicates an interstitial keratitis, either past or present, while if it is on the surface it is an infiltrate, a cicatrix, or a developmental fault. Such congenital opacities afford a great variety of forms and locations, may remain stationary, clear up in time, or increase in size and density.

WOUNDS, FOREIGN BODIES, AND ABRASIONS OF THE CORNEA

A **wound** of the cornea that has invaded the deeper tissues presents a swollen margin and is recognized easily, as a rule. When it has passed through the cornea we nearly always find the iris or the lens to be implicated.

A feeling of annoyance, that may amount to sharp pain, which appears suddenly with photophobia and lacrimation, is quite apt to suggest to the patient that a small **foreign body** has lodged on, or is scraping the surface of his eye, and we are likely to find the substance either entangled in the epithelium of the surface of the cornea, or driven deeper into the tissue. In most cases it is plainly visible as a little black spot, but sometimes it is so small, or is composed of such a translucent material, that we cannot see it with the unaided eye and have to employ oblique illumination and a magnifying glass to locate it. The instillation of a drop of

fluorescin solution into the conjunctival sac is of great help, and by its assistance we may be able to detect a minute abrasion when a foreign body has been dislodged.

Assistance from the same drug may be needed to enable us to see an **abrasion** produced by a twig, or very often by a baby's fingernail. Usually the pain, photophobia, and lacrimation are quite pronounced in these cases, and frequently we can see the abrasion by the light reflected from its edges without the use of a stain, but there is no infiltration unless the place has become infected. In case of infection an ulcer appears which may become purulent if the nutrition of the patient is poor; this is the danger to be feared in a nursing mother, or it may prove the start of a serpiginous ulcer in an elderly person. Ordinarily the lesion heals within a few days, but treatment should be prolonged until the healing is firm, for the reformed epithelium is apt to be attached so loosely to Bowman's membrane as to be detached again by insignificant causes and produce a recurrence of the same condition, or a bullous keratitis, weeks, months, or years afterward, although no new injury is known to have occurred. This is called a **recurrent erosion.**

BURNS OF THE CORNEA

Burns are recognized easily from the great irritation of the eye, the dull, cloudy, perhaps excoriated condition of part or all of the cornea, and the history of an accident in which some alkali, acid, or hot substance struck the surface of the eye. The conjunctiva is red and chemotic, and particles of metal, lime, or other corrosive substance may be found in the sac. Lime burns are the most common. The results depend on the depth to which the tissue is invaded, and the situation of the lesion on the cornea. A deep burn causes an ulcer which may or may not become infected, but heals like all others with the formation of cicatricial tissue. Superficial burns that affect only the epithelium cause sharp pain and photophobia, with a conjunctival redness of the eye, which pass away when the epithelium is restored. The history of exposure to a flash of blinding light, or of prolonged exposure to very strong light, or of the entrance of an acrid liquid or vapor into the eyes, enables us to make the diagnosis.

ULCERS OF THE CORNEA

An ulcer of the cornea is a loss of substance that extends from the surface more or less deeply into the parenchyma, and is caused by a necrosis of the affected portion of tissue. It always starts as a grayish spot with its surface flush with that of the cornea, over which the epithelium may be unbroken, though usually it is steamy and lusterless, in an eye that presents a ciliary injection. This spot is a collection of cells which is called an **infiltrate.** The same name is applied to similar collections of cells that produce a cloudy appearance about the margin, or in the base of an ulcer while it is active; the absence of such infiltrates as these later on shows that the ulcer is healing, or has become indolent.

Several **varieties** of ulcers of the cornea are met with which differ not only in their clinical appearances, but also in their nature and course. Some are peculiar to certain diseases, others are common to many. They may be single or multiple, may attack the surface only, or penetrate deeply into the parenchyma, and may be round, oval, crescentic, or arborescent in form. Vascularity may or may not be a prominent feature. Usually they form grayish or yellowish opacities, some with, others without a purulent infiltration of the margins, walls, or bases. Some of the accompanying symptoms, such as ciliary injection, pain in the eye and brow, iritis, and hypopyon, are proportionate to the seriousness of the condition, while others, like photophobia, lacrimation, and blepharospasm, may be disproportionately severe in mild cases and practically absent in bad ones, so that they cannot be relied upon for information as to the severity of the trouble.

A **simple ulcer** is small, superficial, grayish, with only a slight ciliary injection. Sometimes its margins are slightly infiltrated, and it may show a little tendency to enlarge, when the ciliary injection becomes more pronounced. Occasionally it is somewhat vascular. Photophobia, lacrimation and blepharospasm are apt to be out of proportion to the pain, while iritis and hypopyon are absent. It may be caused by traumatism, and is met with in many diseases, in which it may be multiple, and it frequently forms the starting point from which other varieties develop. In the absence of such a change, and in a well nourished patient, healing takes place quickly, as a rule.

In a badly nourished patient a simple may change into a **deep, purulent ulcer,** which presents a small, round excavation with yellowish walls surrounded by a zone of hazy cornea. The patients complain of pain in the eye and brow, though photophobia is less constant than in the simple variety, the ciliary injection is greater, the conjunctival vessels are congested, and sometimes an iritis with a small hypopyon is excited. This ulcer tends to perforate the cornea, but not to spread laterally.

A purulent ulcer that tends to progress rapidly over the surface of the cornea probably belongs to the extremely virulent **serpiginous** variety; it is met with only in elderly people.

A simple round ulcer that starts near the margin of the cornea and moves toward the center, leaving a groove in which lies a leash of blood vessels, is called **migratory.** Such a condition, which is also called fascicular keratitis, is met with almost exclusively in phlyctenular keratitis.

One or more **crescentic** ulcers sometimes form near the margin of the cornea in severe cases of catarrhal or purulent conjunctivitis, and extend rapidly, following the course of the limbus. The conjunctiva usually is chemotic, and is apt to overhang the corneal limbus so as to conceal the ulcer, which consequently may be overlooked until it is too late to save the eye, unless the overhanging conjunctiva be pushed aside so as to expose the margin as a part of the routine examination when such a chemosis is present. This complication of acute catarrhal conjunctivitis is most apt to occur in patients who are old or debilitated, and it is the less common form of ulceration met with in gonorrheal conjunctivitis of both adults and infants. Sometimes a crescentic ulcer is formed in phlyctenular keratitis by the coalescence of a number of marginal lesions. Unless arrested a crescentic ulcer threatens to cut off the nutrition from the central part of the cornea, which will then break down into pus.

A central infiltrate or ulcer having ill defined edges, with a ciliary injection, but with no pain, photophobia, or lacrimation, suggests a **neuroparalytic keratitis.**

An infiltrate or ulcer in the lower part of the cornea, limited above by a horizontal line, with the adjoining conjunctiva on each side swollen and covered by crusts that may lie partly on the cornea, is a symptom of **keratitis e lagophthalmo.**

A horizontally oval ulcer near the center of the cornea, and on

the line of demarcation of a pannus which covers the upper part, is **trachomatous.**

A chronic ulcer with undermined edges that cicatrizes, recurs with an extension of its margin into the clear tissue, and slowly extends in this way over the surface of the cornea, probably is the rare variety commonly known as **Mooren's.**

A superficial, narrow, zigzag opacity with ciliary injection, photophobia, blepharospasm, and lacrimation, probably indicates a **dendritic keratitis.**

A round depression in the surface of the cornea with walls and base which are slightly hazy or perfectly transparent, associated with no ciliary injection or any other sign of irritation of the eye, is an **indolent ulcer.** It may remain superficial and apparently unchanged for a long time, it may fill up gradually, or it may deepen slowly and even perforate the cornea, if it is left alone. Such ulcers occur sometimes in chronic catarrhal conjunctivitis and in trachoma.

A congenital deep opacity associated with enlargement and ectasia of the cornea, is thought by some to indicate the presence of an excavation or **ulcer on the posterior surface.**

Nebula, Macula, Leucoma

All ulcers and wounds leave cicatrices when they heal that correspond in location, size, and shape to the lesions which produced them, and generally they are proportionate in their density to the depth to which the tissue was invaded. A very superficial ulcer may leave a scar that is so faint as to be scarcely perceptible; this is called a **nebula.** Another that penetrates somewhat more deeply leaves a distinctly visible, though fairly transparent opacity termed a **macula.** One that penetrates still more deeply leaves an absolutely opaque white spot known as a **leucoma.** Nebulæ and maculæ acquired in early life tend to clear up as the patient grows older, so that after some years they may become hard to detect, and impair the vision mainly through the irregular astigmatism they occasion, but this tendency to clear up is much less marked in later life. All signs of irritation leave the eye with their formation, so when we see a spot on the cornea with no vascularity and no ciliary injection we think at once of a cicatrix. Sometimes the cicatrization stops before the loss of substance has been quite replaced and leaves

a concave spot called a **facet.** The only conditions we have to exclude when we see such a spot are congenital opacities, the arcus senilis and spots of degeneration.

Arcus Senilis

We see very often an opaque arc or circle separated from the margin by a narrow band of clear cornea. Unless the patient gives a history of a violent inflammation of the eye in years gone by, this marks a hyaline degeneration which appears to be physiological and not to indicate any impairment of nutrition. It never extends toward the center, and does not interfere with the healing of a wound. Usually it occurs in elderly persons, whence it derives its name, but sometimes it is seen in young ones. Rarely it may be changed into a grooved depression. The presence of a clear zone between it and the margin serves to exclude an opacity left by a deep marginal keratitis in the great majority of cases, though it may not always be possible to differentiate it positively from a scar left by a rather slight crescentic ulcer, when it is known that the eye has been severely inflamed in the long distant past.

Degenerative Changes in the Cornea

A band of opacity that develops quietly, with no sign of irritation of the eye, that extends horizontally across the cornea, and has a peculiar granular appearance that contrasts with the smooth surface of a cicatrix, probably is the result of a calcareous degeneration in which lime salts have been deposited beneath the epithelium. This is the **ribbon shaped degeneration,** which occurs generally in eyes which have been blinded by iridocyclitis or glaucoma, but a few cases of this nature have been met with in eyes that apparently were otherwise healthy. Removal of the salts has produced good results in these last cases.

We may find the vision impaired by the presence of a group of little grayish, subepithelial nodules in the center of the cornea, which change very slowly, are covered by intact and lustrous epithelium, and are accompanied by slight if any signs of irritation of the eye. This is **Groenouw's nodular degeneration** of the cornea. The nodules are situated in and about Bowman's membrane, do not seem to penetrate deeply, and may contain hyalin. Between them a fine grayish stippling of the cornea may be seen. The diagnosis is made

from the appearance, the absence of signs of irritation, and the chronic character. It may be made positive by the microscopical examination of an ablated piece of the tissue.

Very recently *Uhthoff* has described what seems to be a variety of the above in which he found little gray, conical formations extending from the subepithelial part of the cornea down deep into the posterior layers, that did not tend to group themselves in the center or in any other one part, and had no stippling of the cornea between them. We know very little about either of these forms of degeneration.

More commonly when we speak of degeneration of the cornea we refer to a **hyaline,** an **amyloid,** or a **calcareous** degeneration of a corneal **cicatrix.** Occasionally we see what looks like a drop of yellow, or yellowish gray substance in a white leucoma, and then we have reason to believe that the scar is undergoing hyaline or amyloid degeneration; which of these it is we cannot tell without excising a piece and testing its chemical reaction. In other cases we see chalky white granules in a leucoma and understand that earthy salts are being deposited. When the extent of such a degeneration as either of these is considerable the epithelium covering it is apt to give way and so create a superficial, but obstinate ulcer over the site of the leucoma.

Keratocele

When an ulcer has eaten away the tissue of the cornea down to Descemet's membrane, we occasionally are able to see a transparent, rounded bleb in the excavation, surrounded by a gray ring. This protrusion is caused by the driving forward of that membrane by the intraocular pressure so as to make it bulge into the opening, and probably occurs in every case in which an ulcer perforates the cornea, but in most cases it is so evanescent that it is not observed. When a keratocele is seen it may be regarded as evidence that Descemet's membrane is unusually resistant, or that the intraocular pressure is low, and we must not despair of being able to prevent its rupture.

Perforation of an Ulcer and its Consequences

The patient feels a sudden pain in his eye, accompanied by a gush of fluid, when the keratocele bursts, and if we see the case at once we find the iris and lens in apposition with the posterior surface of

the cornea. The perforation may close with the formation of a simple leucoma, but this is not common. Still less so is the production of a scar composed of corneal and cicatricial tissue alone, which is so weak as to bulge before the intraocular pressure and form an ectasia. The **iris** is brought into contact with the internal opening and occludes it in most cases. If the opening is small it may remain attached at this point of the posterior surface, and healing take place with the formation of an **anterior synechia.** A larger opening permits the iris to enter, perhaps to prolapse, and then it remains entangled in the cicatrization with the production of an **adherent leucoma.** When a still larger place in the cornea has been destroyed more of the iris prolapses and unites with the remains of the corneal tissue to form a broad cicatrix, which, if sufficiently firm and strong, flattens and may reduce the size of the cornea by its contraction. If the cicatrix is too weak to do this, or to withstand the intraocular pressure, it bulges forward and gives rise to a **staphyloma,** the size of which depends on that of the preceding ulcer.

In other cases the **capsule of the lens** may block the opening, when healing probably will take place with the formation of a simple leucoma of the cornea and an anterior capsular cataract of the lens. When a central perforation occurs in ophthalmia neonatorum while the child is crying or straining, the lens may be extruded through the opening.

A different result seen sometimes after the perforation of an ulcer, as well as after a wound, whether operative or not, is a failure to heal, leaving a **fistula.** The anterior chamber does not refill, the iris and lens remain in apposition with the cornea, or the anterior chamber may refill to a certain degree and a little bleb or keratocele appear and rupture from time to time. This condition is one which is very menacing to the integrity of the eye.

Intraocular hemorrhage sometimes, though rarely, happens when the tension is reduced suddenly by the escape of aqueous in cases of absolute glaucoma, and in patients whose retinal or choroidal vessels are sclerotic. Finally the perforation of an ulcer opens a channel through which ous agents may enter the eye and cause panophthalmitis.

Ectasia of the Cornea

This is a small protrusion of the corneal tissue alone, without involvement of the iris, which occurs very rarely. A congenital

malformation of this nature has been met with, in which a portion of the perfectly clear cornea projected forward with a different curvature and refraction from that of the rest. The acquired condition appears when the cornea, or a portion of it, has been so weakened by disease that it yields before the intraocular pressure, and has been met with after interstitial keratitis, trachoma and ulcers.

Staphyloma of the Cornea

When a portion, or the whole of the cornea has been replaced by an opaque, protuberant mass, the question is whether we have to deal with a tumor or a staphyloma. The differentiation is easy in most cases, though sometimes it is not, and then we have to rely on the history, which is characteristic in each case. When we are told of a violent inflammation that left the eye practically blind through the production of a corneal scar that gradually grew more and more prominent, the case is one of staphyloma, while if a small growth appeared and grew larger with no preceding inflammation, or if the first symptom was blindness, followed after a time by intense pain in the eye, which was relieved by the appearance of the mass, we know it to be a **tumor.** A staphyloma of the cornea is a cicatrix made up of the remains of corneal tissue, iris, and connective tissue, which has been pouched out before the intraocular pressure, and is traversed by large blood vessels that come from the conjunctiva. If it is of recent formation it is given a bluish color, interspersed with gray and black spots, by the pigment of the iris, and its wall is thin, but as it grows older its wall becomes thicker, firmer, and grayer, so that in an old staphyloma it is thick and white with some dark spots. When large it is apt to overhang the sclera. In rare cases we find an excrescence with a hard, dense outer layer, that has formed on its surface and produced what has been called a corneal horn.

A staphyloma of the entire cornea is called **total;** one that involves only a portion is known as **partial.** A partial staphyloma almost invariably is situated near the margin, leaving a considerable part of the cornea more or less clear, through which a tensely stretched iris can perhaps be seen. No anterior chamber can exist where the cornea is staphylomatous, but a shallow one is present elsewhere, a matter of great importance as it renders possible an iridectomy, by which we may be able to check the progress of the staphyloma and occasionally to restore some slight degree of vision.

After a staphyloma has reached a certain size it will either remain stationary, or rupture. A rupture may heal for a time, but will recur repeatedly until infection enters and destroys the eye. When it does not rupture secondary glaucoma is likely to supervene; then the entire eyeball may become distended, or another staphyloma may form in the sclera.

A small staphyloma may be quite hard to distinguish from an unusually large **cyst** of the cornea, especially when the latter is complicated by iris adherent to the site of the wound, as in a case which was reported by *Collins.*

Ulcers of the Cornea in Infancy

The commonest cause of such a trouble as this in a baby is **gonorrheal conjunctivitis.** The ulcer may be simple, or round and purulent near the center, less often crescentic near the margin. A central ulcer may heal and leave a scar that varies in density from a delicate nebula to an opaque, white leucoma, but, as a rule, it penetrates deeply and perforates if it is not checked. When perforation occurs the iris may prolapse into the opening, and an adherent leucoma be formed, which is likely to develop into a large or small staphyloma; or the lens may come in contact with the cornea with the consequent production of an anterior capsular cataract; or infectious agents may pass from the conjunctiva through the opening and destroy the eye. If the perforation takes place while the child is straining or crying, the lens may be extruded through the aperture. A crescentic ulcer eats away a circle about the periphery of the cornea and the first intimation we receive of its presence, when it has been hidden by the overhanging conjunctiva, may be the sudden breaking down of the central portion from lack of nutrition. The diagnosis of an infiltrate or ulcer in a case of ophthalmia neonatorum is easy, as a rule, but sometimes a bit of pus or mucus that adheres to the cornea leads us into error, especially when the light is not good.

The center of the cornea may be seen to melt away from lack of nutrition in **marasmic** infants, who commonly have xerosis of the conjunctiva. An ulcer may be caused by traumatism, but this is seldom serious unless the child is much debilitated. Occasionally one develops in conjunctivitis due to other organisms than the gonococcus, but such cases are not common. The least rare perhaps are those caused by the diphtheria bacillus, and the pneumococcus.

Ulcers of the Cornea in Childhood

During childhood we occasionally see an ulcer caused by trachoma, gonorrheal or diphtheritic inflammation, traumatism, or as an accompaniment of xerosis in malnutrition, but in the great majority of cases an ulcer is a symptom of phlyctenular keratitis.

Phlyctenular Keratitis

When we see a child seek the dark corners of a room, or bury his face for hours in a pillow, we may be almost certain that he has phlyctenular keratitis. This disease is the same as phlyctenular conjunctivitis, but the symptoms undergo a radical change as soon as the cornea is involved. Photophobia appears, and is so intense that even the light which passes through the closed lids is very painful. Often the blepharospasm is too great to be overcome without the aid of a retractor, while the tears excoriate the skin of the lids, particularly at the outer canthus, where fissures are very apt to be formed. These little fissures increase the blepharospasm, which presses the lids tightly against the cornea and aggravates its condition, thus completing a vicious circle. In a great many cases the lesions to be found on the cornea are much less serious than the violence of these symptoms would lead us to expect.

Many of these children have eczema about the nares, or elsewhere on the face, so many indeed that a large number of ophthalmologists believe this to be an **eczematous keratoconjunctivitis.** Many of them are anæmic, have enlarged cervical glands, and present either the sluggish or the nervous type of **scrofula;** they are either coarse, bloated, with pale skins, thick noses and lips, and slow mentality, or are of slight build, with pale skins that flush readily, little subcutaneous fat, and quick mentality. They are apt to have adenoids, enlarged tonsils, chronic rhinitis, and inflammation of the middle ear. The resemblance to tuberculosis is striking, and while some authorities maintain that scrofula is a distinct disease, "a disturbance of nutrition with a special tendency to inflammation of the lymphatic system," to use the words ascribed to *Grawitz* by *Roemer,* which often becomes complicated by tuberculosis, others assert that there is no such condition as scrofula, and that the entire picture is that of **tuberculous** disease. Without going into the merits of this controversy, which would not exist if either side had proved its case, it suffices to say that the proportion of patients with these char-

acteristics is so great that the disease has received the name of scrofulous keratoconjunctivitis, and that many modern writers believe it to be a manifestation of tuberculosis. The evidence presented by the latter is not convincing, because no tubercle bacilli can be obtained from the ocular lesions, the tissues fail to convey tuberculosis experimentally, while von Pirquet's test, upon which much reliance has been placed by the advocates of this theory, is quite apt to be positive in older children whether they have phlyctenular trouble or not, and does not prove such trouble to be tuberculous even when tuberculosis is present elsewhere in the body. In a minority of cases, though the aggregate number is large, the patients exhibit no other signs than this of scrofula or tuberculosis, and are well nourished, but have alimentary troubles. As alimentary troubles are common in the ill nourished it is a question whether this ocular disease may not be a manifestation of intestinal intoxication in the young. Until some more cogent evidence in favor of one ætiology or another is brought forward, we seem compelled to retain the old term phlyctenular, though this is not satisfactory as it likewise refers to an erroneous idea of the nature of the lesion.

When we open the eye of a child who is suffering thus intensely we find the eyeball quite red, as both the episcleral and conjunctival vessels are engorged, and sometimes a little mucopurulent discharge. About the limbus we may find some phlyctenules, but we shall almost surely see one or more **ulcers** on the cornea, for the symptoms are not apt to be so severe while the phlyctenules remain intact. The ulcer, or ulcers, may be simple, deep, or migrating. Quite a number of phlyctenules and simple ulcers may form an arc not far from the limbus, constituting a marginal keratitis, and these may fuse into a crescentic ulcer. In other cases we may find a multitude of minute ulcers scattered over the surface of the cornea, or the latter may be covered by a pannus, that is distinguished easily from the trachomatous by the entrance into it of vessels from all directions and the absence of a horizontal line of demarcation.

The intense photophobia and blepharospasm, the phlyctenules when they are not yet broken down, and the ulcers usually render the diagnosis easy in a child. In an adult we may not feel quite so certain, unless phlyctenules or scars from other ulcers are to be seen, for this disease seldom appears in an adult who has not suffered from similar attacks during childhood. A simple ulcer may be caused by traumatism, or some other disease, but the history or

the presence of other disease helps us. A migratory ulcer cannot be mistaken for a pterygium if ordinary care is exercised in the examination, and it resembles nothing else, except perhaps a very rare keratitis caused by the aspergillus fumigatus. The small, round, deep ulcer near the margin of the cornea is rarely if ever seen, except in badly nourished persons who are suffering from this disease; the purulent ulcers that accompany the various forms of acute conjunctivitis are likely to be larger and more central if round, and to be crescentic when they are situated near the margin.

Ulcers of the Cornea in Middle Life and Old Age

The only varieties of ulcer that appear characteristically in advanced life are the rare one named for *Mooren,* and the serpiginous.

Mooren's Ulcer

Very rarely in a patient who complains of a chronic inflammation of his eyes, we may see at some point in its course a superficial ulcer of the cornea with an undermined edge, which has started usually near the upper limbus. Vessels extend into it and cicatrization takes place, but sooner or later a recurrence appears in the adjacent clear cornea, more of the surface is made cicatricial, and this continues until the entire surface has been covered and the eye has been rendered blind. This variety seems to have been described first by *Bowman* in 1849, but was named rodent ulcer by *Mooren* in 1867, and since then has commonly borne the name of the latter writer. The disease frequently is bilateral, and it is accustomed to run a slow, but very obstinate course.

Serpiginous Ulcer

When an elderly patient complains of a sudden onset of pain in the eye, brow, and side of the head, which is worse at night, and we find some œdema of the upper lid, a marked ciliary injection, and a yellowish gray, disklike ulcer with an infiltrated, yellowish margin, one part of which is particularly elevated and opaque, surrounded by a gray zone, and accompanied by an iritis and a hypopyon, the diagnosis of a serpiginous ulcer is easy.

A more thorough examination is likely to reveal that the center of the floor of the ulcer is clearer than the periphery, that the yellowish elevation is just outside of the margin beneath the epithelium,

nearly or quite surrounding the ulcer, though more marked at one place, and that fine striæ extend from the hazy zone far into the clear cornea. The most elevated and opaque portion of the yellowish margin is where the active agents are grouped in the greatest numbers, and is called the arc of progression, or of propagation. The faint whitish lines in the clear cornea are formed by wandering leucocytes that have gathered for the defense of the tissue.

The infective agent in the great majority of cases is the **pneumococcus;** *Roemer* found it to be such in ninety-five per cent. Hence this is called sometimes the pneumococcal ulcer. But other agents occasionally cause ulcers that present similar clinical characteristics, so a bacteriological examination is necessary, not simply for an academic diagnosis, but because treatment calculated to arrest the course of a pneumococcal ulcer aggravates one due to **diplobacilli,** and the former thrives luxuriantly on treatment curative of the latter.

It is a curious fact that although pneumococci often are found in the corneal ulcers of childhood and youth, they do not give rise to this variety, which is met with almost exclusively in middle aged or elderly farmers, miners, and other laborers who are exposed to slight traumatisms of the eyes.

The infection always enters the cornea through a traumatic lesion, even though the traumatism may be so slight as to pass unnoticed, for the pneumococcus cannot penetrate or injure the epithelium. A grayish infiltrate, often with one or more yellowish spots near its margin, is formed first, then the epithelium is cast off, leaving an ulcer. The ciliary injection is slight at the beginning, but increases as the ulcer progresses, and is very great when the iritis sets in. At a certain stage in the development of the ulcer the toxic products of the pneumococci become diffused through the aqueous, where they excite an iritis with a hypopyon that may fill a large part of the anterior chamber.

A chronic catarrhal conjunctivitis is present almost invariably, as well as a **dacryocystitis** in the majority of cases. The latter provides an abundant and unfailing supply of pneumococci to roam over the surface of the eyeball, ready to take advantage of the least break in the epithelium and to attack the subepithelial tissue of the cornea. Therefore we should investigate the condition of the lacrimal sac whenever we have to deal with an ulcer of this nature, or with any abrasion of the cornea in an elderly patient. Treatment can

hardly be expected to be very efficient when such a fertile source is allowed to flood the lesion continually with innumerable pneumococci.

The course of the disease depends on the virulence of the microorganisms and the resisting power of the tissues. In rare cases its progress is arrested during the stage of infiltration, when recovery takes place with a scar that is nearly transparent, but, as a rule, the ulcer spreads rapidly over a large part of the surface of the cornea, eats pretty deeply into the parenchyma, and leaves behind a large, dense, white cicatrix which presents an almost insuperable obstacle to vision. Attempts to restore the sight by means of implantation of transparent cornea have succeeded only in very rare instances. When the course is very malignant the entire cornea is eaten away and the eyeball is destroyed by suppuration of its contents.

Ulcers that resemble the pneumococcal closely may be produced by the diplobacillus of Morax-Axenfeld, less often by the streptococcus, the bacillus pyocyaneus, the pneumobacillus, and possibly by the staphylococcus and other microorganisms. The only safe method of **differentiation** is by a bacteriological examination, yet we can obtain some slight assistance from a few clinical facts.

The arc of progression seldom is present, and when existing is less plainly marked, if pneumococci are absent, but considerable past experience is needed to enable the observer to appreciate the difference in some cases. A better point is that in the other varieties the floor does not exhibit the tolerably clear spaces to be seen in that of the pneumococcal ulcer, but is infiltrated uniformly. This may fail in the case of the pneumobacillus, for *Axenfeld* tells us that this microorganism can produce an ulcer which resembles the pneumococcal in every clinical respect. When we have to deal with an ulcer that looks like the pneumococcal, except that its floor is infiltrated evenly, and no bacteriological examination is possible, we may sometimes make a differentiation through a free instillation of a solution of zinc every hour for a day or so, watching its effect closely; if improvement begins, or if the ulcer remains stationary, the diplobacillus is most likely the active agent, while if it continues to progress, this organism is excluded with probability, though not with certainty. The ulcer caused by the bacillus pyocyaneus is characterized by the greenish yellow color of the pus. It is apt to follow the same course as the most malignant form of the pneumococcal, and to result in a rapid destruction of the cornea followed by panophthalmitis.

Ulcers and Inflammations of the Cornea Met with at All Ages

Traumatism may cause a superficial or deep loss of substance, which heals kindly, as a rule, unless it becomes infected. While infection in middle aged or elderly persons is apt to make this the starting point of a serpiginous ulcer, in the young it is more likely to give rise to a round, deep, purulent ulcer. Other ulcers are excited without traumatism by the entrance of fungi and other microorganisms into the tissue, by drying of the epithelium of the cornea, and by lesions that are characteristic of certain diseases or morbid conditions.

Ulcers Due to Fungi

These ulcers are rarely seen and the descriptions given do not agree in all particulars. Few have had the opportunity to see more than two or three cases, many of us have seen none. The disease is said to set in with great, constant, and increasing pain in the eye, so intense as to be out of proportion to the lesions which can be seen at first. A black, grayish, or yellowish white spot appears in the substance of the cornea, surrounded by a circular opacity that breaks down into an ulcer about a central mass which has a dry, or greasy appearance. Iritis and hypopyon appear about this time. Finally the mass is sloughed off and the ulcer heals, leaving a leucoma, though it is quite possible for the eye to be lost through sloughing of the cornea, perforation, and panophthalmitis, if the mass is not removed. A positive diagnosis of the nature of the disease can be made only with the microscope. The aspergillus fumigatus seems to be the commonest agent, but a number of other fungi have been found in more or less isolated cases.

Keratitis e Lagophthalmo

When a patient cannot close his eye so as to cover the entire surface of the eyeball, and presents a reddened, swollen conjunctiva, which is covered with a tenacious mucus, or crusts, where it is exposed, together with an opacity or ulcer in the lower part of the cornea, we have to deal with a condition produced by desiccation and called keratitis e lagophthalmo. The conjunctival mucus is apt to dry and form crusts on the exposed part of the cornea, and when we remove these we find the surface beneath to be dry, lusterless, gray-

ish, and slightly depressed. If the exposure continues the opacity becomes denser, fissures appear in the epithelium, which afford entrance to pyogenic agents, the surface sloughs off, and a purulent ulcer is formed which is bounded above by the horizontal line that marks the lower limit of the surface kept moist by the lid. We find associated with this condition a marked ciliary injection, and very often an iritis with a small hypopyon.

The failure to close the eye properly is due frequently to a **facial paresis,** and then we need to learn whether this is a temporary, or a permanent condition, for guidance in our choice of treatment. One of the things we wish to determine from the associated symptoms is whether the paresis is due to a nuclear, supranuclear, cortical, subcortical, or peripheral lesion. Generally the rule holds fairly good that nuclear and supranuclear lesions produce permanent paralyses, while cortical and subcortical ones produce temporary effects, so far as the twigs of the nerve that supply the muscles about the eye are concerned, and that peripheral pareses may be either permanent or temporary. A peripheral paralysis may be caused by exposure to cold and pass away sooner or later, or it may be due to trouble in the middle ear, elsewhere in the temporal bone, at the base of the skull, or about the parotid gland, when its duration will depend on the curability of the lesion. If galvanism gives the reaction of degeneration we may expect the paralysis to be permanent.

In other cases the closure of the eye is prevented by **mechanical obstacles,** which are plainly evident. Among these may be enumerated exophthalmos, a protruding staphyloma or tumor of the eyeball, a lid that is too short, either congenitally or as the result of traumatism, and an ectropion.

The lids may fail to close and the act of winking be abolished by the benumbing of the sensorium in **coma.** The patient lies with his eyes partially open, oblivious to the irritation produced by the drying and ulceration of the lower parts of his corneæ. Winking becomes reestablished if the patient regains consciousness, and then the keratitis improves at once, but a permanent scar will remain if the epithelium has been cast off. In grave cases of exhausting disease the question is likely to arise whether the condition present is not one of keratomalacia, but the differentiation is easy. In **keratomalacia** the entire cornea becomes hazy and melts away, while in keratitis e lagophthalmo the opacity and ulcer are confined to the lower part of the cornea, and are delimited above by the lower edge of the eyelid.

Neuroparalytic Keratitis

Our attention may be called to an opacity in the center of the cornea, with some ciliary injection, but usually no pain, photophobia, or lacrimation, in a patient who is suffering from some grave lesion of the central nervous system. Closer examination reveals that the surface of the eyeball is anæsthetic, that the secretion of tears is abnormally slight, and we recognize the case to be one of neuroparalytic keratitis.

The picture presented at first is that of a faint opacity in the center of the cornea, which steadily grows more dense, while the epithelium over it loses its luster and finally falls off, leaving an ulcer that soon becomes infected. A ciliary injection appears soon after the commencement of the trouble, but the patient may be annoyed in no way except through the interference with his vision. The purulent ulcer deepens, iritis with hypopyon appears, and perforation may take place with the final formation of a flat, broad, adherent leucoma. This is not the invariable result, for occasionally we may see the dry, lusterless epithelium remain in place. The course is very slow.

Whether the lesion in the cornea is due to a trophic disturbance, or to the irritation produced by dryness dependent on the diminished lacrimal secretion and the lodgment of minute foreign bodies on its surface, which remain undetected because of the absence of sensation, is a question which is still in dispute. None of the arguments advanced on either side are very cogent, perhaps the best is that true trophic nerves have not been found in the cornea, and it is quite possible that the blame should be given to both causes.

It is a matter of much more importance that we should locate the lesion responsible for the corneal anæsthesia through a paralysis of the ophthalmic branch of the trigeminus. If the skin and mucous membranes supplied by all of the branches of the fifth nerve are anæsthetic, the nerve is compromised by a lesion situated at or above the Gasserian ganglion, while if the anæsthesia is confined to the areas supplied by the first, or ophthalmic branch, it may be caused by a lesion anywhere along the entire course of the nerve which interrupts the conductivity of these particular fibers.

When such an anæsthesia immediately follows a **wound,** or a **fracture of the skull,** the cause is evident, the nerve has been divided. When the onset is preceded by neuralgic pain with hyper-

æsthesia or paræsthesia of the skin supplied by the nerve, the patient may be suffering from a **meningitis,** or from a lesion in the **pons, or cerebellum.** The last situation is suggested if the anæsthesia is confined to the cornea and conjunctiva, or if only isolated patches of the skin are affected at the same time, though it is possible for a similar condition of anæsthesia to be produced by a peripheral lesion of the nerve, like an opticociliary neurotomy, or neurectomy. A **tumor** at the base of the brain, in the hypophysis, or in any place where it may compromise the nerve, must be located by means of the accompanying bodily symptoms. When both traumatism and tumor can be excluded **syphilis** is the most likely cause, acting through either a meningitis, or a gumma somewhere along the course of the nerve. Severe neuralgic pain coexisting with anæsthesia, the so-called **anæsthesia dolorosa,** indicates the presence of a lesion that has destroyed the conductivity of the peripheral portion of the nerve, but continues to irritate the distal portion in which the conductivity remains good.

The prognosis of the keratitis depends on that of the cause of the paralysis of the trigeminus. The condition is differentiated from both keratitis e lagophthalmo and keratomalacia by an anæsthesia of the cornea and conjunctiva, which evidently is not dependent on a benumbing of the sensorium, by symptoms of trouble in the central nervous system, and from the former by ability to close the eye.

Dendritic Keratitis

The presence of one or more linear, branching opacities of the cornea associated with pain, or the sensation of a foreign body in the eye, photophobia, lacrimation, and a ciliary injection, is diagnostic of a dendritic keratitis. The signs of irritation vary a great deal in intensity, sometimes they are slight, sometimes they are quite severe, but when they are absent the opacity is cicatricial and is indicative of a former attack of this disease.

We know little of the cause of dendritic keratitis. Its clinical characteristics lead us to believe it to be mycotic, but the agent has not yet been discovered. It is met with at all ages, in both sexes, often after attacks of bronchitis, pneumonia, and other affections of the respiratory tract, and is believed by *Fuchs* and others to be a variety of **febrile herpes.** It is associated so often with malaria as to have acquired the name malarial keratitis, but it has been met

with in connection with scrofula, tuberculosis, and syphilis, and has been known to occur in persons who were apparently healthy. The cornea is apt to be less sensitive than normal, and this together with the fact that a trigeminal neuralgia sometimes precedes or accompanies an attack, raises the question whether the disease may not be connected with an affection of the trigeminus, or the ciliary ganglion, at least in some cases.

The **symptoms** frequently, but not always, begin with a supra-orbital neuralgia, which is followed soon by pain in the eye, photophobia, and lacrimation, caused by the development of a number of small, round elevations of the corneal epithelium. These elevations disappear within twenty-four hours, leaving branched, linear ulcers with little swellings at the ends of the branches. The ulcers extend in lines, which jut out irregularly in various directions, and form grayish furrows that may be invisible for considerable distances beneath the epithelium. If we run a fine pointed probe along the visible grooves, we are able often to plow up others that lead off into apparently healthy tissue, and the disease is apt to be obstinate under treatment until this has been done. The ulceration never passes through Bowman's membrane, and it is rarely, if ever, complicated by an iritis or a hypopyon. The course is likely to be slow, but healing takes place finally with the formation of characteristic opacities that are not very dense.

Febrile Herpes of the Cornea

A number of perfectly clear vesicles, each about as large as a pinhead, occasionally appear on the cornea of a patient who is recovering from some acute febrile disease, and break down into little, round, superficial ulcers, accompanied by a moderate ciliary injection, photophobia, and lacrimation. The ulcers heal quickly, unless they become infected, or the patient is much debilitated. The little vesicles of this febrile, or simple herpes can scarcely be mistaken for anything else. Unlike phlyctenules they are as clear as water and are never vascular, they lack the violent irritation of the eye met with in herpes zoster and in bullous keratitis, and they are much smaller than the bullæ of the latter.

Usually the vesicle is very fragile, so that often we are able to see only its remains in the form of little shreds of epithelium, but occasionally it may be fairly resistant and take a rather atypical

course, as in a case that came under my observation recently. An elderly man, recovering from a febrile disease of his respiratory organs, had a number of minute vesicles as clear as water appear on his left cornea, with little or no photophobia or other sign of irritation, except a slight ciliary injection. They seemed to be either quite transient and to appear in successive crops, or to shift their positions beneath the epithelium, as their arrangement varied from day to day, while their number did not change materially. When one was ruptured artificially a clear fluid escaped and little shreds of epithelium were left hanging, but these soon disappeared. None were observed to become cloudy, and no ulcer could be found at any time, but all disappeared in about a week and left no traces. The resemblance they bore to lymphectasiæ of the conjunctiva was very striking.

Herpes Zoster Ophthalmicus

The eye is affected in only about a third of the cases of herpes zoster, and in these the nature of the involvement may vary a good deal. The conjunctiva is red and chemotic, as a rule, but it is exceptional for vesicles or ulcers to form upon it. The cornea is more or less anæsthetic and may present one or more small, clear vesicles which become cloudy and rupture, so as to give rise to simple or purulent ulcers, or infiltrates may form that are either deep or superficial, diffuse or punctate. Sometimes there is simply a cloudiness or stippling of its surface. Scleritis is seen occasionally. Iritis is not common; when it occurs we usually, though not always, find it associated with a keratitis. Iridocyclitis and choroiditis are still more rare complications. Optic neuritis and atrophy have been observed even when the eye was not affected otherwise. Palsies of the various extrinsic muscles are met with sometimes, and the eye has been known to be lost through panophthalmitis.

When a patient who is suffering from an intense neuralgia presents the characteristic eruption of herpes zoster on the skin of the forehead and lid, together with one or more vesicles, ulcers, or opacities on the cornea, and the usual signs of irritation of the eye, the diagnosis is easy. The only question that can arise is in the case of a central ulcer or opacity, whether it may not be due to a **neuroparalytic keratitis,** which sometimes complicates this disease. A positive answer is not always possible, but in general

it may be said that if the ulcer or infiltrate appeared early it is probably a symptom of the disease itself, while if it appeared late it is more likely to be due to neuroparalytic keratitis. A punctate superficial opacity that has existed almost from the start may break down late and give rise to an infected ulcer, but the differentiation in such a case is of minor importance, as the course, prognosis, and treatment would be the same.

In a small minority of the cases of herpes zoster the *characteristic eruption does not appear on the skin, the lesions are confined to the surface of the eye,* and then the diagnosis may be very difficult. When a violent neuralgia precedes the appearance of vesicles on the cornea, which are associated with a ciliary injection, perhaps congestion and chemosis of the conjunctiva, anæsthesia of the surface, and subnormal intraocular tension, we are justified in a tentative diagnosis of herpes zoster. The vesicles of simple herpes are preceded by a febrile disease; if neuralgia is present it is not severe, anæsthesia of the surface is not marked, and the intraocular tension is normal. The bullous keratitis of glaucoma may be accompanied by pain that radiates over the side of the head and simulates trigeminal neuralgia, and by anæsthesia of the cornea, but the bullæ are much larger, and the tension of the eyeball is much increased. If opacities appear instead of vesicles we must await developments; if they persist for a long time after the subsidence of the neuralgia, and the cornea is anæsthetic, we may make a probable diagnosis of herpes zoster, but a certain degree of doubt is apt to linger in our minds.

Bullous Keratitis

When a patient has a sudden onset of intense pain in the eye and side of the head, with photophobia, lacrimation, and a **large** detachment of the epithelium of the cornea by fluid, which is called a **bulla,** or a large erosion with irregular tags of epithelium hanging from its margins, the first thing we do is to test the tension of the eyeball, for this rare condition occurs most often, perhaps, in old cases of glaucoma. If the tension should be normal or subnormal we would expect to find an iridocyclitis, or some other disease that had affected the eye profoundly, and yet a bulla has been known to appear on a previously healthy eye in a patient who was much debilitated.

If the bulla or erosion is **small,** we question the patient concerning an abrasion which may have happened long before, and we are rather likely to obtain a history of repeated similar attacks, perhaps during a number of years, for this **recurrent erosion,** or traumatic keratalgia, is apt to date back to a simple abrasion, and to recur at intervals of weeks or months for years. It is distinguished readily from simple herpes by the larger size of the lesion and the sharp irritation of the eye, as well as by the history; from herpes zoster by the absence of the other symptoms of this disease; from the other form of bullous keratitis by its smaller size, the otherwise healthy condition of the eye, and the history.

We do not know the cause of the first variety. Possibly it may be found in some interference with the circulation of the lymph which results in an œdema and an accumulation of fluid that detaches the epithelium over a considerable area, but this is hardly a satisfactory explanation. Malaria has been suggested as the cause, but the condition has been met with in cases that gave no history of this, or of any other disease, and no history of traumatism. The cause of the second variety is thought to be too weak an attachment of the newly formed epithelium to Bowman's membrane when the first, traumatic, abrasion healed, so every effort should be made to secure a firm attachment of the epithelium whenever we have to treat even a slight abrasion.

Filamentary Keratitis

It is possible, but highly improbable that we shall meet with a condition in which little globules may be seen hanging down over the cornea, each attached to the latter by a twisted pedicle of epithelium. Each filament consists of cells that have been elongated and flattened into fibrils, twisted together by rubbing on the cornea, and terminates in a knob. Each contains a nerve fibril which renders it very sensitive to the movements of the lids, as well as to the least touch. In addition to this the lesion induces the usual signs of irritation of the eye. Very little more is known about this disease, except that it is said to be chronic and to recur from time to time in old people who are subject to bullous keratitis, or to herpetic eruptions on the cornea, and that it has been known to be caused by wounds.

Superficial Punctate Keratitis

This is the name given by *Fuchs* to gray, punctate, superficial infiltrates scattered over the cornea, associated with an acute conjunctivitis, which usually appear in young people under the same conditions that give rise to febrile herpes, from which it is differentiated by the absence of vesicles. The infiltrates last for a considerable time after the disappearance of the signs of irritation, and then pass away gradually. The formation of an ulcer is a rare exception.

Interstitial Keratitis

When a young person complains of a moderate amount of photophobia and lacrimation, which increase slowly with the progress of the disease, and we find an opacity in the parenchyma of the cornea associated with a more or less marked ciliary injection, we say that he has an interstitial, or a parenchymatous keratitis.

The **clinical appearances** vary considerably in different stages of the disease, and also in different persons, but the following course may be called typical:—A place at the margin of the cornea loses its luster and presents an ill defined cloudiness, which may be seen through a magnifying glass of sufficient power to be composed of many little maculæ situated below the surface. The ciliary injection is slight at this time. After a while similar spots appear elsewhere near the margin; all of these blend and encroach gradually on the center, until the entire cornea may have a bluish gray color, with a steamy or greasy look on the surface, so as to resemble ground glass, due to an uneven condition of the epithelium. During this time the ciliary injection grows more and more pronounced, with the formation of little loops of vessels until the limbus is red and swollen. Other minute blood vessels extend out into the cornea, where they give off penicillate branches. This peculiar branching, their dull, grayish red color, and the fact that they cannot be traced back beneath the limbus to their origins, shows both the vessels themselves and the opacity to be situated deeply in the tissue of the cornea, proves the inflammation to be interstitial, and differentiates the vascularization from that of pannus.

The **vascularization** varies a great deal in different cases. The vessels may be so few and small that we have to search for them, or they may be so abundant as to redden the cornea, or to give it

the yellowish red color known as the salmon patch. At the height of the disease the cornea may be so opaque as to conceal the iris, and the salmon patch may be almost brown red.

In some cases only a portion of the cornea is affected, in some the vascularization appears only in certain parts. If the opacity makes its first appearance in the central part of the cornea the vessels are apt to be few and small. Sometimes the infiltrates remain separate and do not coalesce, a variety that has been called **punctate interstitial keratitis.** Occasionally a ring of infiltration may be seen to form about the center, which remains clear for a long time, and to be separated from the limbus by a fairly clear zone; this has been termed **annular keratitis.** In still other cases the opacities have been known to form a triangle in the lower part of the cornea with its base upward.

One variety that needs to be considered separately, as it is possibly a distinct disease, is the so-called **disklike keratitis.** In this the complaint of the patient is that a spot has appeared recently in his eye and impairs his vision. If we find a gray, diskshaped opacity in the parenchyma of the cornea with a particularly dense place in its center, while the eye exhibits only slight signs of irritation, we inquire concerning some slight traumatism that was received perhaps weeks before, as well as concerning the possibility of a vaccine infection, which has been known to induce this trouble in several instances. Sometimes we find it hard to tell whether the lesion is superficial or deep. The epithelium is dotted all over the surface and may be broken in the center, but ulceration almost never occurs. The surface of the cornea is more or less anæsthetic over the infiltrate, but not elsewhere. With a sufficiently strong magnifying lens radiating, cloudy lines can be seen extending out from the center, which suggest the presence of a colony of some fungus, but the cause is unknown. The condition is differentiated from that due to fungi by the absence of intense pain, and the absence of an ulcer about a central mass. Apparently it is caused by an infection which is not virulent enough to produce suppuration. Relapses are not infrequent, and the trouble may be complicated by the presence of a small hypopyon. The course of the disease is very slow; months may elapse before the opacity begins to clear up about its periphery, and it is quite apt not to disappear altogether. The marked central point of opacity, the radiating lines, the anæsthesia over the infiltrate, and the fact that the affection usually is

confined to one eye, serve to differentiate this from other forms of interstitial keratitis, while an abscess of the cornea is excluded by the absence of violent symptoms and the chronic course.

Interstitial keratitis appears, as a rule, first in one eye and later in the other, although in some cases it attacks both simultaneously. The interval between the affection of the first eye and the involvement of the second usually is short, though it may be several weeks or months. The patients generally are unhealthy children or youths between the ages of six and twenty, mostly between ten and eighteen, though the disease sometimes is present congenitally, and occasionally is seen in older persons. In the last it is apt to run a milder and atypical course.

The name keratitis is somewhat misleading, for the inflammation really is one of the uveal tract and properly should be called a **uveitis.** It is only in the very mildest cases that the iris is simply hyperæmic, iritis is present almost invariably, and we are apt to find evidences of a choroiditis when an opportunity is afforded for an ophthalmoscopic examination early in the disease. The intraocular tension is rather subnormal, as a rule, though it may become increased, and glaucoma has been known to supervene.

The **course** of the disease is slow. The inflammatory symptoms increase for a month or two, their severity somewhat proportionate to the vascularization, and then abate gradually. After the disease has passed its acme the cornea begins to regain its transparency near the margin, and then by degrees toward the center, but several months, perhaps more than a year, are likely to elapse before the tissue attains its final degree of clearness. Good vision is regained in the majority of cases in spite of the fact that hazy patches are apt to persist in the tissue, but the vision remains impaired if one of these patches should happen to be in the center. Years after the attack we may be able to trace the empty blood vessels as white threads in the tissue, if we examine it with a + 16 to a + 20 D lens in the ophthalmoscope, and the presence of these lines is regarded as a positive proof that the eye has suffered at some former time from an interstitial keratitis with a considerable vascularization.

Ulceration of the cornea is very rare, much more so than would be expected when we take into account the bad physical condition of many of the patients. Suppuration has been known to take place in the parenchyma and to form an abscess in a few cases,

and the presence of this disease does not protect the cornea against an intercurrent ulcer induced by any of the ordinary causes, but it may be stated positively that interstitial keratitis does not as such induce ulceration, or any gross prominences on the surface. Yet the epithelium is uneven, as is shown by the reflection of Placido's disk from the surface of the cornea, in which the circles are ill defined but are not distorted. Sometimes a portion or all of the tissue of the cornea is softened and caused to protrude as an ectasia, but much more often serious injury is inflicted by the iridocyclitis, which may lead to the formation of posterior synechiæ, exclusion and occlusion of the pupil, secondary glaucoma, flattening of the cornea, or atrophy of the eyeball.

Most of these cases are due to **hereditary syphilis.** Many of the patients present the physiognomy described by *Hutchinson* as characteristic, "of which a coarse, flabby skin, pits and scars on the face and forehead, cicatrices of old fissures at the angles of the mouth, a sunken bridge to the nose, and a set of permanent teeth peculiar for their smallness, bad color, and the vertically notched edges of the central upper incisors, are the most striking characters." ***Hutchinson's teeth*** have been accepted as nearly diagnostic of hereditary syphilis, but the name does not include all forms of deformed or defective teeth. The two middle upper incisors of the second dentition are small, have lateral margins that converge downward instead of being parallel, and their cutting edges are hollowed into crescents which become deeper as the child grows older, and can be demonstrated until about the age of twenty or twenty-five, when the projecting corners break off, or crumble away. Other symptoms of hereditary syphilis are a labyrinthine deafness, not one due to middle ear disease, large, hard, and painless cervical lymphatic glands, and hard painless nodes on the bones. It is not necessary for all of these symptoms to be present, the combination of a few is sufficient for the diagnosis. The combination of the characteristic teeth, interstitial keratitis, and labyrinthine deafness, which is known as ***Hutchinson's triad,*** is accepted as positive proof of the presence of hereditary syphilis.

In older patients interstitial keratitis occasionally appears as a late secondary manifestation of **acquired** syphilis. In these cases the disease is likely to follow a milder and more rapid course, the cornea is more apt to clear up perfectly, and sometimes only one eye is attacked.

Next to syphilis **tuberculosis** is the most common cause of interstitial keratitis. The presence of tuberculous lesions elsewhere in the body increases the probability that the ocular trouble is of the same nature, but when no clinical signs of either syphilis or tuberculosis can be detected, and the Wassermann test is negative, a ***tuberculin test*** should be made. *Calmette's* method of instillation of tuberculin into the conjunctival sac should never be employed, for it may excite a severe reaction and do grave injury. *Von Pirquet's* test is so sensitive that it is almost always positive in adults and older children whether they have active tuberculosis or not, for the reaction will take place if there is even an extinct tuberculous lesion anywhere in the body, and therefore a negative result is practically the only valuable one, as this may fairly be held to exclude tuberculosis, while a positive reaction needs to be confirmed by the subcutaneous test. The last-mentioned method seems to be the most generally useful, for if it causes a local reaction to take place in the eye, either alone or in connection with a general one, we have good reason to believe the lesion to be tuberculous; if, however, only a general reaction follows in which the eye is not affected, we are still in doubt. The assumption that the lesion is tuberculous is justified in cases in which there is no local reaction, only when no signs of any disease that can excite an interstitial keratitis can be found, and no other tuberculous lesion can be detected anywhere in the body to explain the general reaction.

A tuberculous interstitial keratitis cannot be differentiated from a syphilitic by the appearance of the eye alone, yet a few points occasionally serve to guide our suspicions. The suggestion has been made that the infiltrates seem to be more apt to be distributed irregularly in the tuberculous form; that sometimes they increase in one place while they are clearing in another; that great vascularization is absent, as a rule; that the inflammation is more subject to relapses; and that it is more likely to be confined to one eye; so the observation of any of these things may lead us occasionally to revise our diagnosis, for it is quite possible that a tuberculous interstitial keratitis should develop in a child suffering from hereditary syphilis.

A few cases of interstitial keratitis are due to **local diseases,** like herpes zoster, and in these we may confidently expect the trouble to be confined to one eye. Traumatism is responsible for

a certain number, and attempts have been made to demonstrate this to be a more common cause than is generally supposed, but we still believe that in the great majority of cases it is the consequence, or a symptom, of some general disease. It occurs in leprosy, and has been ascribed by some writers to rickets and scrofula in children, to rheumatism, malaria, influenza, autointoxication, and the climacteric in older patients; but these should never be considered until the ordinary causes, syphilis and tuberculosis, have been excluded beyond the possibility of question. *Arnold Knapp* has reported a case in which interstitial keratitis followed the instillation of a solution of tuberculin in an apparently healthy eye.

Abscess of the Cornea

When a patient complains of very severe pain and photophobia, and presents some œdema of the lids, an intense injection of both the ciliary and the conjunctival vessels, and a cloudy cornea with a central grayish or yellowish disklike opacity, the periphery of which is the most opaque, over which the surface is depressed but not broken, together with a violent iritis and hypopyon, he has an abscess of the cornea, sometimes called an **onyx.** The course of such an abscess is violent and usually terminates in rupture with the formation of an ulcer, probably serpiginous in character. Less often recovery takes place with the absorption of the pus and the production of a dense cicatrix. This resembles somewhat the appearance described under interstitial keratitis as disklike keratitis, but can hardly be mistaken for it. In an abscess the symptoms of inflammation are violent and the course is rapid; in disklike keratitis the subjective symptoms are slight, mainly impairment of vision, and the course is very chronic. A less important point, as a rule, is that the periphery of the opacity is the most opaque portion in the former, while the center is in the latter. An abscess is likewise distinguished from an infiltrate from any other cause by the intensity of the inflammatory symptoms and the depression of the surface of the cornea. It can hardly be mistaken for an ulcer caused by fungi, because the latter has a central point about which an ulcer forms.

The cause is infection with pus agents that have entered either from without through a wound that has healed over superficially, or from within by metastasis. It may follow a contusion, an abra-

sion, a lacerated wound, or an operation, especially in an unhealthy patient, or it may appear in the course of some acute infectious disease, like smallpox, typhoid fever, measles, or scarlet fever. Cases have been met with in which the symptoms of inflammation were slight or absent, and as some of them occurred in unhealthy children they were suspected to be tuberculous.

Striped Keratitis

Sometimes we see a number of **white lines** appear very deeply in the cornea after a wound, usually after a cataract extraction. According to *Nuel's* observations, confirmed by *Hess* and others, these lines are formed by a wrinkling of Descemet's membrane and, properly speaking, are not indicative of a keratitis, although there is some œdema in the deep layers of the corneal tissue. The only subjective symptom is a temporary impairment of the vision, which soon passes away as the tissue regains its transparency. Such a striate opacity is not an indication of danger, does no harm, and usually clear up in a few days or a few weeks.

A similar wrinkling of Descemet's membrane has been observed in some cases of detachment of the retina and in other conditions in which the tension of the eye was very subnormal. Permanent striated opacities that present the same appearance may be produced by bichloride of mercury, when a solution of this drug has been used to irrigate the anterior chamber after operation.

Deep Punctate Keratitis

This name has been applied to the **deposit of bits of fibrin** on Descemet's membrane, but this condition is symptomatic of cyclitis and not a keratitis at all. Quite a number of brown dots are to be seen congregated, more or less in the form of a triangle, on the posterior surface of the lower quadrant of the cornea in the majority of cases, but occasionally the dots are larger, fewer, and whitish in color, when they are called by English writers "mutton fat" deposits.

Sclerosing Keratitis

During an attack of grave inflammation of the eye characterized by a livid discoloration and bulging of the sclera in the neighborhood of the cornea, the sclera may seem to encroach on the cornea by the

formation of an opacity that extends over the limbus toward the center. This opacity is broadest at the limbus, generally is irregular in shape, though sometimes it has a fairly curved, indistinct edge, and it causes the margin of the cornea to have a jagged appearance. It is of a dull white or yellowish color, is situated in the parenchyma, and is a symptom of deep scleritis. After the inflammation has subsided the opacity may clear up partially, but in most cases it changes to a tendinous, bluish white, and is permanent.

KERATOMALACIA

When we see an extremely emaciated patient who has xerosis of the conjunctiva, and a more or less grayish or bluish, insensitive cornea, perhaps covered with scales, we know that this tissue is in imminent danger of breaking down into a large purulent ulcer and melting away. This keratomalacia is a bad sign for the life of the patient. It is met with at all ages, but most often shortly before death in **marasmic children** who are suffering from cholera infantum. In older persons it may appear late in the course of such exhausting diseases as dysentery, typhoid fever, puerperal fever, cholera, or scarlet fever, and may be produced by starvation. As a rule, the patients are in a stuporous condition.

Keratomalacia is the breaking down of the cornea because of insufficient nutrition. Infection with pus agents takes place secondarily. The only diseases with which it is in danger of being confounded are keratitis e lagophthalmo and neuroparalytic keratitis. The former sometimes appears in the same exhausting diseases, but it is to be distinguished by the absence of xerosis, the presence of reddened, swollen conjunctiva in the region of the palpebral fissure, and the location of the lesion in the lower portion of the cornea, while in keratomalacia the entire tissue is involved. Neuroparalytic keratitis appears in patients who present symptoms referable to a lesion of the central nervous system, rather than to the diseases mentioned, and presents a central opacity, or a round, purulent ulcer.

TUMORS OF THE CORNEA

A tumor may be situated on the surface of the cornea, or it may have burst through from within the eye; in either case it is met with rarely. A superficial tumor ordinarily starts from the conjunctiva,

though a few have been reported from time to time as primary growths of the cornea itself. It may be an epithelioma, a sarcoma, a myxoma, a fibroma, a papilloma, a keloid, or a dermoid, but it is seldom that the clinical characteristics are such as to enable us to determine the nature of a growth without a microscopical examination. Cysts seldom are large enough to be seen, but occasionally they cause slight protrusions. A congenital growth with a dry, downy or hairy surface, usually at the corneoscleral junction, is a dermoid. All of the rest start, as a rule, near the margin of the cornea as little spots that gradually increase in size until they impair the vision. Sometimes a portion of one can be lifted up so as to expose the cornea beneath it; in other cases this cannot be done, because the tissue itself has been invaded. As a rule, a space can be found where the cornea is sufficiently clear to allow us to make out an anterior chamber of ordinary depth, and an iris that is not tightly stretched. When a pigmented, vascular tumor has invaded the entire cornea we may have to depend on the history to differentiate it from a staphyloma, but it is not very often that we have any difficulty in recognizing a tumor to be such.

When an intraocular tumor has burst through the cornea we have a history of blindness that came on before any external lesion became apparent, followed later by glaucomatous pain, which persisted while the eyeball became enlarged and deformed by a protrusion of the cornea, and ceased, or at least was much relieved, coincidentally with the rupture of the tumor through the coat of the eye. We always find the shape of the eyeball to be distorted otherwise as well, and the freed tumor grows rapidly.

CHAPTER IX

The Sclera and the Anterior Chamber

THE SCLERA

It is not common to find anything of pathological interest on the sclera, aside from the injection of the minute episcleral vessels about the cornea in inflammations of that tissue, or of the iris and ciliary body, and the engorgement of the anterior ciliary veins when the eye is suffering from glaucoma, but occasionally we see scleral conditions that are of more or less importance. These include spots, protrusions, and inflammations.

PIGMENTATIONS OF THE SCLERA

A congenital, irregular, brown patch is to be seen on the sclera, quite frequently in negroes, much less often in brunette whites, which is of no pathological signification. Rare cases of pigmentation in Addison's disease have been reported, and these sometimes are situated symmetrically in both eyes near the cornea. Little black or brownish stains may indicate where grains of powder have been driven into the tissue through the conjunctiva, and silver has been held responsible for a bluish stain in a few cases. Of much greater importance is a roundish, slate-gray, or blackish spot, which is accompanied by a history of a past inflammation of the eye that resisted treatment for a considerable time; such a spot may be single, or a number of them may form an arc or a circle about the cornea, when we know that the eye has suffered from recurrent attacks of scleritis, each of which has left a cicatrix.

STAPHYLOMA OF THE SCLERA

A protrusion outward of some portion of the sclera, with few or no signs of inflammation of the eye, is a staphyloma. The only ones that are visible externally are those situated in front of the

equator of the eye, which are called anterior, and those about the equator itself, which are known as equatorial. When a portion of the back part of the sclera bulges outward to form a **posterior staphyloma,** we can sometimes make out a localized concavity in the fundus with the ophthalmoscope; this will be discussed under the lesions of the fundus. An **anterior** or **equatorial staphyloma** is dark because the sclera at this point has been so thinned as to allow the color of the subjacent uvea to become visible. An anterior staphyloma over the ciliary body is known as **ciliary,** one between this place and the cornea as **intercalary.** Sometimes an intercalary staphyloma involves both the sclera and the cornea.

A staphyloma is caused by a local weakness of the sclera that renders it unable to withstand the intraocular pressure. When the latter is above normal in a young child the entire sclera is apt to stretch, but in older persons it is more likely to give way in places. The cause of the weakness in the posterior segment that gives rise to a posterior staphyloma is unknown, and that which operates in the anterior segment cannot always be determined positively, but in the great majority of cases it is the thinning of the tissue produced by disease or traumatism. Occasionally we see one or more anterior or equatorial staphylomata in old cases of absolute glaucoma, or one caused either by the imperfect cicatrization of a wound, or by the erosion produced by a tumor, but usually an anterior staphyloma bears witness to a past scleritis. This is particularly true when several have been formed about the cornea, or when the latter has been projected forward by the yielding of a dark band which surrounds it in the sclera. A ciliary, or an intercalary staphyloma is a serious menace to the eye, as it is very apt to induce secondary glaucoma.

SCLERITIS

When we see a dark red, or bluish-red protrusion of the sclera, with a rather indistinct margin, associated with a violent iridocyclitis and its usual subjective symptoms, we have to deal with a **deep scleritis.** Both eyes are apt to be affected, and we may be able to see opacities in the vitreous, or masses of pigment in the fundus that attest the presence of a choroiditis. Sometimes a sclerosing keratitis is present, or several dark spots may be visible about the cornea, which show where one scleritic node has succeeded

another. Occasionally there is a uniform swelling about the cornea instead of isolated prominences.

The combination of iridocyclitis with an angry swelling over the site of the ciliary body, to which the conjunctiva is not adherent, makes the diagnosis of this rare disease fairly easy. Whether it appears primarily in the sclera and spreads to the uvea, or starts in the ciliary body and involves the sclera secondarily, is still an open question. The result is apt to be destruction of the eye, whether the tissue of the sclera is thinned so as to give rise to the dark cicatrices or staphylomata already described, or the sclera is enormously thickened, as in the variety known as brawny scleritis.

Brawny, annular, or **gelatinous scleritis** appears typically as a gelatinous, brownish red infiltration of the conjunctival, episcleral, and scleral tissues, most marked close to the limbus. The succulent swelling often overhangs the cornea, and extends back a variable distance, to the equator or farther. The blood vessels and lymphatics are dilated enormously. Pain is distressing and tenderness is acute. A sclerosing keratitis, accompanied by both deep and superficial blood vessels, advances until it has covered the entire cornea, or the changes may be most marked in the posterior segment of the globe. The progress of this disease is extremely slow, and in its early stage we are quite likely not to be able to distinguish it from the ordinary variety of scleritis. The prognosis is bad; the blind eye usually has to be enucleated on account of pain. One eye alone has suffered in most cases, but in three that have been reported both eyes were practically blinded, and in two others the second eye was affected mildly.

Little is known of the causation of scleritis. It has been ascribed at various times to rheumatism, gout, tuberculosis, gonorrhea by metastasis, syphilis, and menstrual disorders, but in most cases we are not able to find a definite cause. It is ordinarily thought to occur mainly in adults who are younger than those who are victims to episcleritis, but *Derby* found that ten of the sixteen pathologically examined cases of brawny scleritis occurred in patients between sixty and eighty years of age, the youngest was thirty-four, the oldest seventy-six. Children do not seem to be subject to scleritis.

Episcleritis

When we see a circumscribed, reddish, elevated spot with a rather yellowish center and a lilac or violet periphery, which appeared suddenly, the question is whether it is a **phlyctenule,** or a nodule produced by an **episcleritis.** In neither case is there apt to be much irritation of the eye, photophobia, lacrimation, or any circumcorneal injection, yet, as a rule, the differentiation is easy. Phlyctenular conjunctivitis is seldom seen except in childhood, while episcleritis is a disease of adult life. A phlyctenule causes no pain and is not tender, while an episcleritis is apt to be attended by a dull pain, and the affected part of the globe is tender to the touch. The conjunctiva immediately surrounding it is hyperæmic in either case, but if we move it back and forth we can see that a phlyctenule and all of the redness moves with it, while an episcleral nodule remains fixed, together with the lilac or violet vessels, and allows the conjunctiva to slide over it. A drop of adrenalin solution blanches the conjunctival vessels quickly, but does not affect the deep, episcleral ones so readily. If there have been former attacks a history of obstinacy, and the presence of one or more grayish spots in the sclera, may decide the question in favor of episcleritis. A phlyctenule is transient and soon breaks down, while an episcleral nodule seldom changes after the first day or two, but persists for weeks and never ulcerates. The nodule finally becomes absorbed, and either leaves no trace, or a little gray spot, this depending on the depth to which the sclera has been invaded. Recurrence is the rule, but the same spot is never attacked twice.

Episcleritis varies in severity and, though it does not induce the destructive lesions characteristic of deep scleritis, it may be attended by iritis, or by a sclerosing keratitis when it occurs close to the cornea. Sometimes a slight, diffuse episcleritis is to be observed in interstitial keratitis.

The cause of a typical recurrent episcleritis is obscure. It is most apt to be met with in middle aged or elderly people, though occasionally it is seen in younger adults, and frequently is ascribed to rheumatism or gout. Sometimes arteriosclerosis, chronic nephritis, and diabetes have been believed to be responsible. Similar nodules are to be seen in leprosy, and occasionally in syphilis, but they are not apt to recur in these diseases. Primary tuberculosis of the sclera is questionable, according to *Parsons,* though the disease may extend to it from the uvea.

Episcleritis Periodica Fugax

Occasionally we meet with a case in which the patient has a circumscribed deep hyperæmia of the episcleral vessels, over which the conjunctiva is congested, so that a little red spot is formed on the white of the eye, which is not associated with any noticeable swelling, lasts a few days, and recurs at intervals. Such a spot with such a history marks the mildest form of episcleritis. It is differentiated readily from a subconjunctival hemorrhage by the presence in it of distinct vessels, and from a phlyctenule by the congestion of the episcleral vessels and the absence of any little nodule.

THE ANTERIOR CHAMBER

As we glance through the cornea we need to note the depth of the anterior chamber, any irregularities that may be present, the condition of the aqueous, and any collection of pus, blood, or exudate that it may contain. An adhesion of the iris to the cornea will cause one part of the chamber to be shallow, but when one part is much shallower than another, and this is not accounted for by such an adhesion of the iris, we look at once at the pupil, for if this is large and oval we have good reason to fear that a transparent lens has been dislocated partially into the anterior chamber. An isolated shallow place may indicate the presence of an exudate, a tumor, or a foreign body in or behind the iris. If a circular protrusion of iris surrounds the pupil, while the margins of the latter are bound down to the anterior capsule of the lens, we have a crater pupil, which will be described later.

We can perceive at once a very great deviation from the normal depth of the anterior chamber, but sometimes it is difficult to be sure of a slight, or even of a moderate variation. We must not pronounce upon it too quickly, for the depth of the chamber varies physiologically, not only with the size and length of the eyeball, but also at different periods of life in the same individual. It is shallow in early life, deeper in the adult, and becomes shallow again as age advances, but practically it is always the same in the two eyes, unless these differ widely in other respects. When only one eye is diseased a comparison of the two by oblique illumination will inform us in most cases whether its anterior chamber is ab-

normally shallow or deep, but when both eyes are affected we may still be in doubt.

Obliteration of the Anterior Chamber

When the iris and lens are in apposition with the posterior surface of the cornea, or are separated from it by so minute a quantity of aqueous that it cannot be perceived, we say that the anterior chamber is absent, or has been obliterated. Unless the cause is plain we test the tension at once, for if this is subnormal we may be sure that an **aperture** exists through which the aqueous is escaping, while if it is above normal it is evident that the iris and lens have been driven forward by pressure within the eye itself.

An external aperture may have been formed by a wound, or by the perforation of an ulcer, when we do not have to search for the cause, as a rule. Yet sometimes we find an empty anterior chamber in a patient who gives a history of a sudden blurring of vision, perhaps after a sharp pain in the eye, but none of any lasting pain, traumatism, operation, or severe inflammation, and then it will not be surprising if we find that an indolent ulcer has perforated. It is also possible, though by no means common, for a wound to have evacuated the chamber without attracting the attention of the patient in any other way. An anterior chamber that remains empty for more than a few hours indicates that healing of the opening is delayed and that a fistula is being formed. Sometimes we meet with such a delay after a cataract extraction, or an iridectomy, and then it commonly is due to the presence in the wound of a bit of iris, anterior capsule, or fibrin, which holds the lips of the wound apart. In other cases an anterior chamber may be seen to reform partially and then to disappear again repeatedly; the cause of this is that the aperture closes with a cicatrix which is so feeble that it ruptures as soon as the intraocular tension has reached a certain height.

Shallow Anterior Chamber

An anterior chamber that persists in remaining shallow after a cataract extraction, or an iridectomy, when the wound shows no delay in union, probably indicates a detachment of the choroid, which will soon be replaced. If it is associated with an œdematous swelling of the conjunctiva about the wound, the cicatrix contains a

fistula through which aqueous is escaping into the subconjunctival tissue. Ordinarily we find the tension subnormal in both of these cases, but should it be normal or elevated, we inquire into the possibility that a trephine operation has been performed for glaucoma.

When the **tension is above normal** we find glaucoma in the majority of cases, but the same condition may be caused by a tumor, an exudate, or a hemorrhage in the back part of the eye. The differentiation has to be made from the history and the accompanying symptoms, and it may be easy or very difficult. When the media are clear we ought to be able to see a glaucomatous excavation of the papilla, and that the other possible causes are absent, or else detect these causes themselves; but when they are not clear the problem is not so simple. A history of blindness that preceded the onset of pain, and a mass behind the lens, may indicate an intraocular growth; a preceding violent iridocyclitis that has blinded the eye and left it painful may lead us to suspect an exudate; a history of traumatism, or the presence of blood in the aqueous may lead us to diagnose a hemorrhage; but the differentiation of an intraocular tumor from glaucoma, or from a postlenticular exudate is by no means always easy. A flattened cornea also accounts for a shallow anterior chamber.

Deep Anterior Chamber

The anterior chamber is rendered unusually deep by such an abnormal curvature of the cornea as we find in keratoconus, keratoglobus, and infantile glaucoma. When we see a deep anterior chamber associated with a tremulous iris, we know that the lens has been either dislocated or removed. A deep anterior chamber associated with a ciliary injection and a number of dots on the lower part of the posterior surface of the cornea is symptomatic of cyclitis.

Visible Changes in the Aqueous

A reddish aqueous, or a **hyphæma,** shows the presence of blood. During a cataract extraction, or an iridectomy, blood may enter the anterior chamber from wounded vessels of the conjunctiva or iris, when it is of little consequence, as a rule, but if the hemorrhage should continue, or if it should recur in company with an iridocyclitis, the prognosis is not very good. Spontaneous hemorrhages

sometimes take place into the anterior chamber in cases of hemorrhagic glaucoma, intraocular tumors, leucocythæmia, purpura, and hæmophilia, and occasionally they accompany the onset of attacks of iritis. Other hyphæmas are produced by traumatism. Whatever may be its cause, if a hyphæma persists for a considerable time, its coloring matter may enter the cornea and cause a blood stain.

A **cloudy aqueous,** or an **exudate** or **hypopyon** at the bottom of the anterior chamber, directs our attention to the iris, whether any other tissue is inflamed or not. Hypopyon is a collection of pus cells which often contains no pyogenic microorganisms, when we have to call it sterile pus. In many cases of keratitis its formation seems to be due to the irritation of the iris by the toxines of the pyogenic organisms in the cornea which have diffused through the aqueous. When it is fluid it is bordered above by a horizontal line, and may be scattered by shaking the head, but when clotted it forms a mass at the bottom of the chamber with a convex upper margin while increasing, and a concave one while passing away.

A growth in the anterior chamber with more or less transparent walls and clear contents is a **cyst of the iris,** which may be small, or so large as to nearly fill the entire chamber. An opaque growth comes from the iris and may be a lymphoma, a gumma, a tubercle, or a sarcoma.

CHAPTER X

The Iris

We need to be familiar with the appearance of the normal iris, and with its muscular movements, if we are to detect with certainty any abnormal deviations. Its surface is lustrous, but uneven; its color varies physiologically from blue to a brownish-black, and it presents near its center a round opening called the pupil, and we have to observe any lack of luster, any pathological unevenness or color, and any fault in the contour, position, or reactions of the pupil.

The pupillary margin has a **black edge,** which is the termination of the retinal layer, and the only part of the central nervous system that can be seen without artificial aid. It appears to be broader when the pupil is contracted, and to become narrower as the latter dilates; it is finely serrated, and sometimes presents a circumscribed black mass that may look like a little tumor, or may overhang the tinted part of the iris so as to counterfeit the appearance of a notch in the margin. When such a mass is congenital and does not increase in size, it is called an ***ectropion of the uvea,*** and is of no pathological importance. Next to this black edge comes the **region of the sphincter,** a narrow circular band with minute radiating folds on its surface that become deeper as the pupil contracts. Next to this is a circular elevation, the **minor circle** or corona, which divides the iris into two unequal parts, the **pupillary zone,** that includes the black edge and the region of the sphincter, and the **ciliary zone,** that occupies the greater part of the visible iris. In the latter we see folds which radiate out from the minor circle, and other lines that run deviously, but in a generally radiating direction, with depressions known as **crypts** between them, which give the iris a mottled appearance through the shadows formed in them, or through an irregular distribution of pigment. In addition to these there are circular furrows that run concentrically to the circumference and grow deeper as the pupil dilates; these are the **contraction furrows** or **folds.** The outermost part of the iris which is hidden

from view by the opaque margin of the cornea is called the **peripheral zone,** and an arterial plexus in its outer edge is known as the **major circle.** The elevations and depressions are symmetrical and form the details of the design of the iris, and any elevation of the surface that is not symmetrical and does not form one of the details of this design is abnormal.

PERSISTENT PUPILLARY MEMBRANE

Occasionally we see an eye in which cords or tags of tissue extend from the anterior surface of the iris partly or wholly across the pupil. When they arise from the corona, or from the ciliary zone, they are remnants of the fetal pupillary membrane, but when they arise from the pupillary zone they are to be looked upon as indicative of some pathological condition. Remnants of the pupillary membrane sometimes extend across the pupil from one point to another on the surface of the iris, always outside of the pupillary zone, and occasionally one can be seen to be attached to the anterior capsule of the lens.

COLOR OF THE IRIS

The color of the normal iris depends not only on the pigment in the retinal layer, but also on the amount of pigment in the chromatophores which lie in the connective tissue of the stroma, and may be blue, gray, hazel, or some shade of brown. When the pigment in the chromatophores is scanty the color of the iris is blue. The iris of an albino appears to be pink when seen by light reflected from the fundus, and blue at other times, because pigment is wanting in both the chromatophores and the retinal layer. The greater the amount of pigment in the chromatophores the darker is the iris, which may be almost black, and the more does its surface have a dusty or granular appearance. The color of the two irides is usually, but not always, the same, yet often we have to rely on a comparison of the two in order to detect a pathological change of color, which may be simply a darkening and readily escape notice. When the iris of a diseased eye is greenish and that of its mate is blue, we infer that the stroma is infiltrated. A muddy, greenish, or brownish color with loss of luster indicates the presence of exudates either on the surface of the iris itself, or on the

posterior surface of the cornea, or else a turbid aqueous, very often any two or all three of these. A yellow-brown, rusty color suggests siderosis, or old, recurrent hemorrhages within the eye. A brassy, or dirty straw color leads us to think of a purulent iritis.

The distribution of pigment is not always even. In many irides there are little dark or light colored spots, aside from the crypts, where the pigment is more or less dense than elsewhere. These **pigmented nævi** are physiological in the great majority of cases, but when a little dark spot in his iris has recently caught the attention of an adult patient we should look to see if it is elevated above the surrounding surface, and try to keep it under observation until we can ascertain whether it is stationary, or is increasing in size, for a melanosarcoma has been known to start in this manner in very rare cases, and to grow for quite a while without causing irritation. In other cases the distribution of the pigment is so irregular that one part of the iris may be blue or gray, while another part is brown, but as long as the normal luster is present we need not think of an atrophy confined to a certain sector. Such a **heterochromia** may exist in one or both eyes. The color of the two irides is altogether different in some cases, and the same name, heterochromia, is applied to this condition. The luster of a light-colored iris is brighter than that of a dark one, but no matter what the color, it is never dull under normal conditions.

THE NORMAL PUPIL

We speak of the pupil as central, though when we wish to describe its location accurately we have to say that it is slightly below and to the inner side of the center of the iris. It is round, has a regular contour, and varies greatly in size under physiological conditions, so much so that figures which purport to give its average diameter are of little practical service. It is smallest, and its margins are quiet, during deep sleep, but if we watch it through a strong magnifying glass during consciousness we shall see that it is never quiet, but makes incessant minute movements to regulate the quantity of light which enters the eye, to shut off diffusion circles that tend to blur the retinal images, and perhaps in response to mental processes. Any change in the light causes a perceptible change in its diameter, and when the light is steady its diameter may vary as much as 2 mm., according to the amount of

accommodation called into action, and is increased greatly by the play of certain emotions. The pupil of a myope is apt to be larger than that of a hypermetrope, but its size varies in both with the age of the patient. It is small in infancy, increases in size till about the age of five, remains at its maximum until about twenty-five, then begins to grow smaller, and is quite small again in old age. This wide physiological variation renders it impossible for us to detect minute abnormal deviations, but we can tell when a pupil is dilated to an unusual extent, or strongly contracted. Abnormal dilatation of the pupil is called **mydriasis,** abnormal contraction **myosis.** In the great majority of people the pupils of the two eyes are practically equal, and a marked difference in size commonly is a pathological symptom, but occasionally we meet with a case in which this **anisocoria** is physiological.

REACTIONS OF THE PUPIL

The contractions and relaxations of the muscles of the iris in response to stimuli make themselves manifest in certain reactions of the pupils, modifications of which are of great diagnostic importance.

Reaction of the Pupil to Light

The pupil contracts when the illumination is increased, and dilates when it is decreased; this is the **direct reflex reaction** to light. Ordinarily we test this reflex by covering both eyes for a short time and suddenly removing the screen from before one of them, or by flashing a stronger light than that of the room into one pupil, which is then seen to contract. The pupil of the other eye contracts at the same time by what is called the **consensual** or **indirect reaction;** this contraction is very slightly less, but practically equal to that produced by the direct reaction.

It is believed at the present time that the light stimulus is received by the rods and cones of the retina, possibly only by those of a rather small area in the region of the macula, transmitted through the coarser fibers of the optic nerve to the chiasm, where part of them decussate, thence along the dorsolateral parts of the optic tracts to the corpora quadrigemina, and then, by a course as yet uncertain, from this point to the nucleus of the sphincter, which forms a part of the oculomotor nucleus. This nervous tract for conveying the stimulus from the rods and cones to the nucleus is

called the **centripetal neuron.** The stimulus for the contraction of the sphincter starts in its nucleus and follows the **centrifugal neuron** in the inner part of the motoroculi into the orbit, through a branch of this nerve to the ciliary ganglion, and in the short ciliary nerves to the iris and the sphincter. The centripetal and centrifugal neurons together form the **light reflex arc.** Probably association fibers exist between the light reflex arcs of the two sides which, together with the centripetal neurons that decussate at the chiasm, account for the consensual reaction, for the pupil of a blind eye may not respond to light directly, and yet light thrown into the other eye may excite a consensual reaction.

Rather less than half a second elapses between the entrance of light into the eye and the beginning of the visible contraction of the pupil, which occupies from three fourths to one second, but the reaction may be rendered slow or slight by a pathological condition, and then it may be of considerable diagnostic importance to determine whether it has been completely abolished or not. When we feel uncertain as to the results of the above test we take the patient into a dark room, let him remain a few minutes until his retinæ have become adapted to darkness, then flash a bright light directly into his eye and watch closely for any movement of the pupil. If there is no movement the reflex is abolished, if there is a slight or slow one it has been impaired.

Reaction of the Pupil to Convergence

If we place an object a few inches in front of a patient's eyes, in the midplane between them, his two internal recti, ciliary muscles, and pupils will contract as he fixes upon it, if they are all in a normal condition. Thus we have an associated convergence, accommodation, and contraction of the pupils when we look at near objects. Whether the accommodation or the convergence is responsible for the condition of the pupil has been the subject of much discussion, but at present its contraction is referred by most students to the convergence. Although it is automatic this reaction is dependent to a certain degree on the will and therefore is not a pure reflex. The centripetal neuron runs from the cortex of the brain to the oculomotor nucleus, the centrifugal through the motoroculi, the ciliary ganglion, and the short ciliary nerves to the sphincter and Mueller's muscle, both of which contract.

Myotonic Convergence Reaction

Once in a great while we meet with a patient in whom the reaction to convergence is slow in one or both eyes, the pupil remains contracted for some time after the convergence has ceased, and then proceeds to dilate slowly. The phenomenon is more noticeable when one eye only is affected. In a case reported by *Oloff* the left pupil contracted slowly while the eyes were fixed on a near object, but finally became smaller than the right; then when the patient gazed into the distance the right pupil dilated promptly, while the left remained contracted for about thirty seconds and finally dilated slowly. The nature of this trouble is uncertain; some observers have thought it to be an incomplete absolute immobility of the pupil, but this opinion has not been accepted. In most cases it is associated with a faulty light reflex, and sometimes the accommodation is paretic, but these conditions are not present in every case. It has been met with after traumatism, and in such diverse diseases as alcoholism, measles, tabes, migraine with attacks of fainting, general paresis, diabetes, multiple sclerosis, exophthalmic goiter, and neurasthenia. Probably it has a central origin, but it is of little or no diagnostic value at present.

Reaction of the Pupils to Closure of the Lids

If we hold the lids apart and the patient tries hard to close the eye, we see the pupil contract, probably because the impulse sent to the muscles through the facial branch of the motoroculi is communicated to the sphincter. This reflex is both direct and indirect, and is to be seen most plainly in cases of reflex immobility of the pupils. It was discovered by *von Graefe,* and has been studied considerably by *Gifford,* but it is of little diagnostic value, except that when we have to deal with a lesion at the oculomotor nucleus, it enables us to learn whether the sphincter is paralyzed or not.

Cortical or Attention Reflex of the Pupils

Haab discovered that when a person's attention is called to a light placed at his side as he looks forward at a dark wall, his pupils will contract and remain small until his attention has been called to something else, although his eyes have not moved and the illumi-

nation has been constant. Other observers have found that the same reflex was obtained when the presence of a light was suggested even though it were absent. The only information gained from this test is that the nucleus of the sphincter and the centrifugal neuron are unimpaired. It has been obtained in eyes that were totally blind from optic atrophy, and in some cases of reflex and total immobility of the pupil.

Reflex Dilatation of the Pupil

It goes without saying that dilatation of the pupil follows all of these reactions as soon as the cause of the contraction ceases to act, but the pupils dilate also in reflex response to sensory and psychic stimuli, as well as to irritation of the sympathetic nerve. Such a **reflex dilatation** is caused by pain, whether internal, like the various forms of colic, or external through irritation of the sensory nerves of the skin, conjunctiva, or cornea. The pupil can be made to dilate slowly and widely by pricking the skin of the malar region for half a minute, and then it contracts suddenly. Strong emotions, like fear and anger, likewise cause the pupils to dilate widely. The dilatation induced by these sensory and psychic stimuli is able to overcome the most powerful contraction that is produced by light. Another dilatation which is considered to be psychic occurs when a person awakes from a sound sleep. During sleep the pupils are very small, but they dilate widely the moment the person wakens, so that he is dazzled if he is in a bright light; if he is not dazzled we are likely to find the pupil in a state of spastic or paralytic myosis, induced perhaps by toxæmia, opium, or morphine.

MALFORMATIONS OF THE PUPIL

In addition to abnormal dilatation and contraction we often see pupils which may be central and oval or irregular, or displaced from the center and more or less distorted, while occasionally we find more than one aperture in the iris, or that a part or the whole of the iris is wanting. Most of these malformations are the results of pathological conditions, but some are congenital, and we need to be able to distinguish these at once.

Posterior Synechiæ

When a central pupil has an irregular contour, or assumes one when it has been dilated, we know that one or more parts of its margin are bound down to the anterior capsule by adhesions which are called posterior synechiæ, and are infallible indications of a present or past iritis. These posterior synechiæ usually form projections of the iris tissue that jut out into the pupil and distort the latter into a great variety of shapes, but occasionally they glue the edge alone along a part of its length to the anterior capsule. The synechiæ may be few or many, and sometimes the entire margin is bound down, when we say that the pupil is excluded, or secluded; in such a condition the aqueous is apt to accumulate in the posterior chamber and bulge forward the ciliary zone of the iris so as to form what is called a crater pupil. In other cases the entire contiguous surfaces of the iris and lens are bound together by a total posterior synechia. When the pupil is filled by an exudate lying on the anterior capsule it is said to be occluded. When any such conditions as these are present at birth they are to be ascribed to an intrauterine inflammation, not to a developmental fault.

Oval Pupil

When a pupil is central, but oval in form we notice its size and the depth of the anterior chamber. If it is large and the anterior chamber is evenly shallow we look for other signs of glaucoma; if it is large and the anterior chamber is unevenly deep, we have good reason to suspect a dislocation of the lens; if it is large and the anterior chamber is normal, the condition may possibly be congenital. If it is small we instill a mydriatic to ascertain whether it is held in this shape by posterior synechiæ, if these are absent we look for evidences of disease in the central nervous system, of which an oval or rather irregular pupil sometimes is symptomatic, and if no indications of inflammation or disease, either past or present, can be found we may conclude it to be congenital.

Corectopia

In rare cases we may see in both eyes an oval or irregular pupil at some distance from the center of the iris, and be told that the malformation has been present since infancy; this is congenital corec-

topia. Other members of the family are apt to have a like defect, as often it is hereditary. Frequently it is associated with a corresponding ectopia of the lens, or with some other abnormality of the eyes. To determine whether it was caused by a fault of development, or by an intrauterine inflammation, we test its reaction to light, for in the former case its reaction to light is likely to be perfect, while in the latter the response is apt to be faulty because of adhesions, or of atrophy of the iris.

As a rule, it is not difficult to differentiate congenital corectopia from a condition in which the pupil has been displaced from the center as the result of inflammatory adhesions of the iris to neighboring structures, such as may follow a wound, or the perforation of an ulcer of the cornea. In most of these cases the pupil is not only drawn to one side, but is much distorted, with a point extending toward the site of the lesion, where we are likely to find a prolapse of the iris, an anterior synechia, or an adherent leucoma. Sometimes the pupil is drawn upward after a cataract extraction, and is not much distorted, while no such adhesion of the iris is visible. The history serves for differentiation.

Polycoria

A few cases of supernumerary pupils have been recorded, but there seems to have been in most of them a more or less regular central pupil with one or more dehiscences in the iris which were not provided with sphincters. The term has been applied erroneously to bridge coloboma, but we mean by polycoria the congenital presence in the iris of two or more apertures which appear to be pupils. A somewhat similar condition can be caused by a gaping wound of the iris, by an iridodialysis, by an iridectomy that leaves the margin of the pupil intact, by the breaking down of a gumma, and by localized patches of atrophy, but the differentiation can be made easily, as a rule, from the history, and the presence of other signs of traumatism or disease. An interesting diagnostic point is that congenital polycoria is said not to interfere with vision, while artificial apertures are apt to cause monocular diplopia.

Iridodonesis

By this term we mean that the iris can be seen to vibrate or tremble when the patient moves his eye. When this tremulousness

is well marked it is evident that the iris is not supported properly by the lens, which must be dislocated or absent, but if we look closely we can see a faint iridodonesis even in a normal eye when the pupil is much contracted. A well marked tremulousness of the iris can be present congenitally only in cases of ectopia or congenital aphakia; as a rule, it indicates that the lens is pathologically shrunken, dislocated, or has been removed.

Iridodialysis

When one side of the pupil forms a straight line we shall probably find a black segment at the corresponding part of the ciliary margin of the iris, where the latter has been torn from its attachments so as to leave an aperture. This separation of the peripheral part of the iris is called an iridodialysis, and is the result of a traumatism, usually of a contusion. When it is small it may cause no trouble, but when it is large enough to act as a second pupil it is apt to cause monocular diplopia. In a very large iridodialysis the detached portion of the iris may be reversed so that we are able to see the black retinal layer; this is called **anteversion** of the iris.

Inversion of the Iris

In very rare cases after a contusion of the eye we may find that a portion of the iris has disappeared because it has been bent backward over the ciliary body. Should the entire iris be inverted in this manner the same appearance is produced as that presented in traumatic irideremia, and the differentiation cannot be made, as a rule, until after the recovery of the eye from the effects of the traumatism. In the majority of cases an inversion of the iris is associated with a dislocation of the lens, and it cannot take place unless the zonule has been ruptured.

Irideremia

A part, or the whole of the ordinarily visible portion of the iris may be absent either congenitally or as the result of traumatism. The differentiation is not very difficult.

Total irideremia, in which no trace of the iris can be seen clinically, is very rare. The entire area covered by the clear cornea presents the solid black of the pupil, which gives a strange, expressionless appearance to the face. With the ophthalmoscope we

obtain the red reflex of the fundus throughout this area, and sometimes we can see the ciliary processes. Most of these cases are congenital, but the entire iris may be torn away as the result of a severe contusion of the eye, and either fall into the anterior chamber as a little, shrivelled, blackish or grayish mass, or be extruded from the eyeball through a laceration in the sclera, perhaps in company with the lens. Accidents have been known to occur in which the entire iris was torn away as the result of a violent movement on the part of the patient, or of an unsteady hand on the part of the operator, when the iris had been seized for a proposed iridectomy, and left in the grip of the forceps outside of the eye. The same thing is done intentionally in the operation of iridenkleisis, which is rarely performed. The appearance after recovery in these cases is much the same as that of congenital irideremia, but the differentiation usually is easy, even when the history is unknown, for traumatic irideremia, or aniridia, nearly always is unilateral, while the congenital variety is almost invariably bilateral. Out of 117 collated cases of the latter there were only two in which the defect was not total in both eyes, and only one patient had a complete iris. In addition to this, when an eye has suffered a traumatism sufficient to produce an avulsion of the iris scars can usually be found. Another point is that photophobia is very pronounced and annoying in traumatic irideremia, if the eye otherwise is good, but seldom is present when the defect is congenital. Out of 164 cases of total and partial congenital irideremia photophobia was said to have been severe in only six, slight in twenty-nine, and quite a number of these patients had corneal troubles sufficient to account for the symptom. The absence of marked photophobia is particularly striking when the vision of the eye is good. Another guide is the position of the lens when it is displaced. A traumatic dislocation may be in any direction, though it is not very apt to be upward, while in congenital irideremia it is common for the lens to become luxated upward gradually, inclining a little perhaps to one side or the other, to shrink, and to become cataractous.

In **partial irideremia** a portion of the iris is wanting, while the rest is visible. Probably it is true that every case of congenital irideremia is partial histologically, as a rudimentary iris has been found in all of the very few cases that have been examined anatomically. *Pagenstecher, Lembeck, Debendetti,* and *Rindfleisch* agree in describing this rudimentary iris as caught between two lamellæ of

Descemet's membrane, but for clinical purposes such an invisible rudiment might just as well be nonexistent.

After two or three broad iridectomies have been performed beside one another in the same eye, the condition of the iris resembles that presented in many cases of congenital partial irideremia, yet it is not hard to distinguish these conditions. In most cases the history informs us aright, but the presence of a disease for which iridectomy is the correct treatment is not a safe guide, because glaucoma may occur in either total or partial irideremia. The presence of a cicatrix in the scleral margin is good presumptive evidence that an operation has been performed, but we can obtain positive proof from an examination of the margins of the defect. If the design of the iris has been cut across and the sphincter is divided, we know that an iridectomy has been performed, for in a congenital defect the sphincter can be traced along its margin, growing narrower as it approaches the periphery, and the details are not interrupted sharply, although they are altered.

The cause of congenital irideremia is unknown further than that it is apt to occur in families which have a hereditary predisposition to anomalies of the iris. In the majority of cases it is associated with developmental faults of other parts of the eye, or of other parts of the body. The ciliary processes, choroid, retina, and optic nerve frequently are defective or diseased, the cornea often is faulty and seems to be unusually prone to inflammation, and the lens is apt to become cataractous and to be gradually luxated upward, even when it appears to be normal at first. In a minority the irideremia seems to be the only defect, except that myopia is quite common, and good vision may be secured by correction of the refractive errors.

Coloboma of the Iris

A congenital cleft of the iris that resembles a Gothic arch with its apex downward, or downward and inward, is a congenital coloboma. When the apex is visible and the fissure evidently does not involve the whole width of the iris the coloboma is partial; when the apex is hidden behind the opaque margin of the cornea it is total. A partial coloboma may vary in size from a notch in the lower margin of the pupil to a cleft which extends nearly to the root of the iris.

A coloboma commonly is supposed to be caused by a faulty closure of the fetal cleft, but the correctness of this theory has been

questioned of late by some embryologists who maintain that the fetal iris has no cleft at any time during its development. If these investigators are right we are just as much in the dark with regard to the origin of coloboma as to that of irideremia. Sometimes it is met with in several members of the same family, so there may be a certain amount of hereditary predisposition, but this is not as marked as it is in many other congenital defects. It is associated frequently with coloboma of the ciliary body and choroid, sometimes of the sheath of the optic nerve, and of the lens, but it is also met with alone. By itself it does not impair the vision or cause photophobia.

A band of tissue passing across the defect from one side to the other converts it into a **bridge coloboma.** A strip of tissue without pigment running downward in the iris is called a **false coloboma.** *Beard* gives the same name to a dark stripe caused by an ectropion of the uvea when this physiological lump of pigment causes the pupil to look as though it were elongated downward. A condition in which the stroma of the iris is absent so as to expose the retinal layer, causes a similar appearance. Ordinarily we speak of the defect produced in the iris by an iridectomy as an **artificial coloboma;** many writers call congenital deficiencies of varying size that extend in any direction from the pupil except downward, **atypical colobomata;** and great confusion exists in literature because of the way in which this term is used. One writer limits the size of a coloboma to one-fourth of the iris and says that it must always extend downward; another says in his description of what he called a coloboma that the entire nasal third of the iris was wanting and that the balance formed a crescent; a third calls a case in which the central, superficial layers of the iris were absent, so as to leave a bright ring of color about a dark membrane which contained a round pupil in its center, an incomplete coloboma. A better name for the last would seem to be either partial superficial irideremia, or congenital exfoliation of the iris. Whether a fissure of the iris that extends downward is due to a faulty closure of the fetal cleft or not, its frequent association with a similar cleft of the choroid and ciliary body marks it as different from like defects elsewhere in the iris, which seldom if ever have similar associations, and entitles it to a distinguishing name. The others can be included properly under the term partial irideremia, or partial aniridia.

IRITIS

When a patient has acute pain which is worse at night, radiating over the brow and temple of one side of the head, extending back perhaps as far as the neck, the diagnosis of supraorbital neuralgia is too apt to be made without even looking at the eye, and to be thought sufficient, especially when it agrees with the patient's own preconceived opinion. Yet just such a pain may be symptomatic of an ulcer in the cornea, an iritis, or a glaucoma, and eyes have been lost from each of these diseases because no attempt was made to ascertain whether the origin of the neuralgic pain was in the eye. In other cases the eyes were thought to be suffering from a slight intercurrent conjunctivitis of no importance, or to be reddened and lacrimating in sympathy with the affection of the trigeminal nerve. It does not seem safe to make any exception to the rule that in every case of severe supraorbital pain the eye should be examined thoroughly enough to exclude any of these diseases. If the lid is swollen and the eyeball reddened at all, we may feel pretty confident of finding the trouble there. An ulcer of the cornea in such a case is plainly visible and can be overlooked only through carelessness or pure neglect. If the cornea, iris, pupil and tension appear to be normal, we must search carefully for a small foreign body as the possible cause, but in the great majority of cases we shall find either a small, irregular pupil, indicative of iritis, or a dilated pupil with increased intraocular tension, equally diagnostic of glaucoma. The differentiation of these two diseases will be given more at length under plastic iritis, and under glaucoma. When we see in the eye of a patient who complains of this neuralgic pain which is worse at night, a swollen upper lid, an extensive, dark episcleral injection, and a discolored, muddy looking iris with a small irregular pupil, the diagnosis of an **acute plastic iritis,** the most common variety, is positive and easily made; but then our work has only just begun—we have to determine its ætiology if possible. Less often we meet with the serous and purulent forms of iritis, and we need to be able to recognize all under different conditions, for any type may run an acute, a subacute, or a chronic course. An iritis that appears as a complication of a severe inflammation of the conjunctiva, cornea, sclera, or of some other part of the uvea is called secondary, all other forms are primary. Occasionally the inflammation may be chronic from

the start, as in some forms of tuberculous iritis, the quiet iritis sometimes met with after operations for cataract or glaucoma, and in plastic iridochoroiditis. The subjective symptoms are pain, photophobia, lacrimation, and impairment of vision. The more acute the attack the more severe the pain, is a rule to which there are exceptions. Photophobia and lacrimation often are not very severe, and in the early stage of a purely chronic iritis the only subjective symptom may be an impairment of vision.

Hyperæmia of the Iris

When we see an iris that is discolored, the hue darkened, or perhaps with a reddish tone when the original color was dark, or greenish when it was blue or gray, the pupil contracted or less responsive than normal to darkness or a mydriatic, but with *no posterior synechiæ,* we say that it is congested or hyperæmic. Sometimes we can see little red lines on its surface which are dilated blood vessels. In other cases we can find nothing wrong with the iris except an imperfect response of the pupil to any mydriatic.

Such a condition may be secondary to inflammation or irritation of nearly related parts of the eye, as when the cornea has been irritated for quite a while by a foreign body, or when the conjunctiva, cornea, or sclera is inflamed; in such a case no further signs of inflammation may develop, and the hyperæmia may disappear with the removal of the exciting irritation, but this does not always happen, and when it does not the hyperæmia proves itself to be what it really is—the first stage of an iritis.

Serous Iritis and Cyclitis

When a patient complains of blurred vision with a sense of discomfort in the eye, which may amount to considerable pain, and we find an œdematous upper lid, a slight rosy, or bluish red ciliary injection, a more or less triangular collection of dots, with the apex of the triangle upward, on the lower part of Descemet's membrane, a deep anterior chamber, and a pupil that may be dilated or normal in size, but responds slowly to light and has a few slight posterior synechiæ, we recognize a **serous iritis.** If there are no posterior synechiæ the case is one of **serous cyclitis** and the pupil is almost surely dilated. The two conditions usually are combined, so the

proper name for most cases is **serous iridocyclitis.** The intraocular tension is raised at first and falls later, but may rise again as a symptom of secondary glaucoma if the excretion of fibrin is increased and extensive posterior synechiæ are formed. The dots on Descemet's membrane usually are minute, brown, and in large numbers, but sometimes they are larger, whitish, fewer, and less easily perceived by the naked eye; the latter are known by English writers as "mutton fat" deposits. The aqueous is cloudy and sometimes a small hypopyon is to be found. If the opacities on the cornea and the cloudiness of the aqueous permit us to see into the eye we are apt to find the vitreous hazy, but occasionally we are able to see buff colored spots in the peripheral parts of the choroid, sometimes with opacities at the posterior pole of the lens, all indicative of choroiditis.

We know very little of the ætiology of this disease. It may usher in a sympathetic ophthalmia, so we should always investigate concerning any previous wound of the other eye. Further than this all we know is that it has been ascribed to nearly if not quite all of the diseases which are provocative of plastic iritis, to malnutrition, anæmia, uterine troubles, and eyestrain, and that it occurs far more commonly among women than among men. It runs a slow course, lasting from six weeks to three months or more, and it may lead to blindness, but the prognosis under proper treatment is pretty good, except when it is of a sympathetic nature, or when it recurs too frequently.

What may possibly be a distinct variety is called by some writers **recurrent chronic cyclitis.** The attacks recur every three to six months for a number of years. The majority of the patients are women who suffer from menstrual troubles, but the disease has been met with in men who had been suffering from nervous exhaustion induced by overwork and great mental anxiety.

Serous is differentiated from plastic iritis by the mildness of the subjective symptoms, the normal or enlarged pupil, the deep anterior chamber, and the scantiness of the posterior synechiæ in its early stage. It may pass over into the plastic form, and this change is shown by the later increase of the synechiæ.

When the pupil is large and the tension plus, the question may arise whether we are not dealing with an inflammatory glaucoma, but an increase of tension enough to render the cornea sufficiently hazy to obscure the iris to the same degree as a turbid aqueous is

attended always by severe pain in the eye and head, a dark engorgement of the anterior ciliary veins, a shallow anterior chamber, and more or less anæsthesia of the cornea, none of which are present in serous iritis or cyclitis. Moreover, the deposits on Descemet's membrane are characteristic and diagnostic of cyclitis.

Plastic Iritis

When a patient suffers from intense pain in the eye, which radiates to the brow and temple, blurred vision, photophobia, and lacrimation, and presents an œdematous upper lid, a darkly reddened eyeball, especially about the cornea, tenderness to pressure over the ciliary region, discoloration and indistinctness of the details of the iris, we may be dealing with a case of **acute plastic iridocyclitis,** ordinarily called iritis, or of **acute glaucoma.** If the pupil is small, sluggish, and irregular, the anterior chamber of normal depth and possibly containing an exudate, the cornea sensitive, and the intraocular tension about normal, the case is one of iridocyclitis; if the pupil is large, oval, and immobile, the anterior chamber shallow, the cornea insensitive, and the tension much increased, it is one of glaucoma.

Sometimes the symptoms are modified in that the lid is not œdematous, the vision is not much blurred, and there is little or no tenderness over the ciliary body, because the inflammation is mainly, if not wholly, confined to the iris. In some cases the symptoms are so very indefinite at first that we may not be certain whether the case is one of conjunctivitis with much congestion but little discharge, or of a commencing iritis; a doubt of this nature may be set at rest by the effect produced by a mydriatic. If the pupil dilates evenly and with normal rapidity in response to the drug the case is one of conjunctivitis; if it dilates evenly but more slowly and to a less extent than it should, the iris is hyperæmic; if the iris is bound down at all to the anterior capsule by posterior synechiæ, iritis is present. Occasionally the tension thus exerted on fresh, weak synechiæ causes them to rupture and allow the pupil to dilate evenly, but little black spots are left on the anterior capsule of the lens which serve equally well for the purpose of diagnosis.

This instillation of a mydriatic for diagnostic purposes is proper in any case of serious doubt, but before we employ it we should always assure ourselves that the pupil is not larger than normal,

and that the tension of the eye is not plus. The best way to test the tension is with the finger tips, for the use of the tonometer for this purpose is likely to excite the patient unnecessarily with no compensating advantage, and is too much like a theatrical procedure to impress the patient with the surgeon's armamentarium. In young persons we usually employ atropine, as the use of this is to be maintained if an iritis is found, but in elderly patients it is better to use cocaine or euphthalmine first, and to change to atropine if the pupil fails to respond normally.

When all of the other symptoms of iritis are pronounced, but the pupil refuses to dilate at all, we shall probably find that the effect of the drug is prevented mechanically by synechiæ that surround and exclude the pupil, or by a total posterior synechiæ, conditions that indicate that the iritis is unusually violent, or else is old and neglected.

At times, though not always, we can see red vessels coursing over the surface of the iris, little circumscribed swellings arising from the stroma, or a hyphæma, a hypopyon, or a grayish collection of fibrin at the bottom of the anterior chamber. These are all diagnostic of iritis when they occur in combination with other symptoms, and usually are so when alone, though the red vessels may be indicative simply of a hyperæmia, a hyphæma may be caused by traumatism, or any intraocular hemorrhage, and a small, circumscribed swelling at the bottom of the anterior chamber may be a tumor, either benign or malignant, which has not yet caused local irritation.

Ætiology of Iritis

A generation ago it did not bother us much to decide what we believed to be the cause of nearly every case of iritis with which we met. If it was neither of traumatic origin, secondary to some other inflammation of the eye, nor a symptom of sympathetic ophthalmia, and did not occur during the course of some such acute infectious disease as measles, we felt confident that it was to be ascribed to either syphilis or rheumatism. Each of these diseases was held to be responsible for about one half of the cases that came before us, although it was acknowledged that there was quite a list of possible causes to which an occasional case might be referred when no indication of either of these could be elicited. This division was held to be so nearly absolute that when an iritis occurred in a young

person it was practically up to him to prove that he did not have syphilis, unless he had a distinct rheumatic history, and when one developed in an elderly patient it was almost certain to be pronounced rheumatic. The diagnosis was based largely on the history, so much so that a very indefinite one of syphilis, or even of known exposure to the disease, or one of obscure rheumatic pains in the joints or muscles, would be accepted as positive evidence of the nature of the lesion. The idea that one of these diseases must be responsible was so pronounced that evidence of a past iritis was accepted as very strong evidence of syphilis, and in 1886 I knew an eminent syphilographer to make a diagnosis of syphilis in a patient who presented no history, and no other sign of the disease except the dilatory healing of the wound after an operation for fistula in ano. In view of the general acceptance of this theory by men of the highest standing so short a time ago it would be a matter of surprise if it did not prevail at the present time in the great bulk of the profession, yet among those who are best informed it is well known that our ideas concerning the ætiology of iritis have undergone a change that seems to be almost revolutionary. Years ago an occasional voice protested against such a haphazard method of diagnosis, and gradually methodical, painstaking investigations of the physical conditions of the patients have been substituted, and these have demonstrated the great prevalence of causes which had been supposed to be exceptional, as well as the fact that a prior infection with syphilis, or the presence of so-called rheumatic pains, does ***not necessarily*** indicate the ætiology in any particular case. The most recent investigations along this line were presented by *Brown* and *Irons* in a paper read before the American Ophthalmological Society in May, 1916. One hundred cases of primary iritis, which were of neither traumatic nor sympathetic origin, were subjected to the most exhaustive scrutiny in search of the cause, and the results were presented in the following table, in which rheumatism is conspicuous by its absence. The figures cannot be accepted as percentages, because of the small number of patients. It is not to be expected that all possible causes should be met with in this number, but the list may fairly be supposed to include the most common ones.

THE CAUSES OF IRITIS IN ONE HUNDRED CASES

Infections	Alone	With Other Infections	Total	Coincident Infections						
				Syphilis	Gonorrhea	Tuberculosis	Dental	Tonsil	Sinus	Genito-
Syphilis	10	13	23	..	8	5	5	1	1	..
Gonococcal infection	7	2	9	..	..	..	1	1	..	..
Tuberculosis	8	..	8	..	..	..	..	..	..	..
Dental infection	7	11	18	2	2	2	..	7	1	..
Tonsillar infection	7	9	16	1	3	2	7	..	..	..
Sinus infection	1	2	3	..	..	..	1	1	..	..
Genito-urinary (nonvenereal)	3	..	3	..	..	..	..	..	..	..
Other infections	2	..	2	..	..	..	1	..	..	..
No cause found	1	..	1	..	..	..	..	..	..	..
Combined infections	..	..	17	8	9	7	8	13	5	1
			100							

Next to the complete absence of rheumatism from this list of diseases perhaps the most striking feature of the above table is the enormous reduction of the proportionate number of cases in which syphilis was the actuating factor. This disease appears as the cause of the iritis in 23 cases, and as a coincident infection in all other instances in which the iritis was traced to other causes, a total of 34. In five other cases the disease had to be taken into account, in three because of a history of infection, three, twenty, and forty-five years previously, in whom the Wassermann test repeatedly proved negative, and who presented no other evidence; and in two because of a one plus Wassermann test with no history, and no other evidence of syphilis. In each of these patients other active infections were present, in one dental, in one sinus, in one tonsillar, in one gonococcal, and in one combined of sinuses and tonsils, while in none of them did the evidence seem to warrant the consideration of syphilis even as a coincident infection. The importance of this stands out clearly when we realize that only a very short time ago not only the twenty-three, but thirty-four or more of these cases would have been diagnosed and treated as syphilitic iritis.

The number of cases in which these writers found ***two or more possible causes*** to be present is surprisingly large. In the majority of them it was possible to determine which was the actuating cause by a careful weighing of the evidence, but in some this could not be

done, and these cases were classed together as due to combined infections. **Apical infections of the teeth,** or alveolar abscesses, were demonstrated in forty-one patients by the X-rays, and this diagnosis was confirmed on extraction of the teeth, but they were regarded as ætiologic in only eighteen. The evidence on which such an infection was decided to be the actual cause included the failure of treatment directed to other possible causes to give relief to the eye, the prompt recovery of the iritis after the abscess had been evacuated, the association of attacks of iritis with those of known dental trouble, and the absence of other causes. **Tonsillar infections** did not include superficial lesions, but only deeply situated abscesses in or about the tonsils, some of which were hard to detect. The evidence on which they, as well as other localized infections, were adjudged to be ætiological was much the same as in the cases of dental abscesses.

The results of the modern researches into the ætiology of iritis, of which the above is simply the latest example, go to prove that while *syphilis remains preeminent* as a cause, the number of cases that can be ascribed to it properly is much smaller than it was thought to be formerly, while the *importance of rheumatism as a factor seems to have nearly reached the vanishing point,* but we must not hurriedly consider the latter to have been eliminated. We have to take into account now as in the past syphilis, gonorrhea, tuberculosis, diabetes, malaria, typhoid fever, pneumonia, all of the acute infectious diseases, as well as gout and rheumatism, but above all we must search out hidden foci of infection in structures distant from the eye, not only in the accessory sinuses, the tonsils, and the roots of the teeth, but also in the prostate, and other organs. The one common factor in all of these seems to be an infection that may be generalized, as in an acute infectious disease, or localized, as in an alveolar abscess. Still we are not in a position to say that such an infection must be present in all cases. Some have been attributed to menstrual troubles, to autointoxication, and to anæmia, and we cannot yet assert that all of these must have been due to unrecognized foci of infection, even though we are ready to admit that many of them were in all probability. This statement is borne out by the fact that *Brown* and *Irons* were *not able to detect any cause* whatever in one of their one hundred cases.

The great difficulty in determining the ætiology seems to be not a paucity of possible causes in the majority of cases, but rather too

great an abundance of adequate ones. We must remember that iritis is not necessarily syphilitic because the patient has been infected with syphilis, nor tuberculous because he is suffering from tuberculosis, any more than it is necessarily malarial because he has had that disease. A patient may suffer at the same time from syphilis, tuberculosis, carious teeth, tonsillitis, and rheumatic pains that may or may not be traceable to an old gonorrhea or gleet, and any of these diseases alone might furnish an adequate explanation. When two or more causes are found to be cooperating in this manner we can eliminate the activity of all but one in many cases, but sometimes we cannot, for when treatment is directed to all of the morbid conditions simultaneously, as should be done always for the best interests of the patient, we are apt to remain in the dark as to the nature of the real cause; or it is possible in such a case that the iritis really is due to a combined infection. Still there are conditions in which a probable diagnosis is made readily. If the patient is in the secondary stage of syphilis, this disease is likely to be found responsible; if he has ever had gonorrhea and is suffering from rheumatic pains in his joints, the iritis is quite apt to prove to be gonorrheal; if he has other symptoms of tuberculosis we may be able to prove this to be the cause through a local reaction to tuberculin. The cause of an iritis may be said to be evident also when it complicates an acute infectious disease, a wound or inflammation of the eye, and to be extremely probable when it sets in some weeks after a wound of the other eye.

No one can differentiate the cause of an iritis from the appearance of the iris, yet occasionally we are able to get some help from an inspection. Sometimes we can see with the aid of a magnifying glass and oblique illumination that the iris seems to be scarcely swollen at all, but shows on its surface red lines, which are blood vessels, together with a grayish deposit of fibrin, perhaps with minute threads floating out from it, which renders the design indistinct, while one or more parts of the dark edge of the pupil appear to be glued to the anterior capsule so as to form curved, linear posterior synechiæ, which are largest and strongest below, with their concavities toward the center of the pupil. In another case the whole iris seems to be swollen and the posterior synechiæ are pointed or angular projections with broad bases into the pupil. It is not very often that we see a case of iritis which presents such characteristics typically, but when we do we are able to draw a distinct inference

as to the nature of the inflammation. In the first case the surface of the iris is the part mainly affected, and the synechiæ have been formed by the settling of fibrin between the contiguous surfaces of the iris and lens; in the second the entire tissue of the iris is involved and the synechiæ have been formed by exudates from focal points of inflammation in the parenchyma of the iris. This differentiation can be made only in a certain number of cases, and, as a rule, only in the first attack of the disease, not only because there are many intermediate grades between the two conditions which modify the clinical appearance, but also because repeated attacks change the picture so much that a clear distinction is impossible. When it can be made it affords a little help toward determining the probable cause, since a superficial iritis seems to be met with chiefly in connection with those diseases and inflammations which are apt to excite rheumatic pains and formerly were grouped together as rheumatic, while a parenchymatous iritis suggests instead a disease that is known to create foci of inflammation in tissues, such as syphilis or tuberculosis. In some cases of syphilitic and tuberculous iritis these foci in the iris become large enough to form visible local elevations, and are then of great help in making the diagnosis.

Secondary Iritis

It would seem as though an iritis which accompanied an ulcer of the cornea, or an acute conjunctivitis, would be recognized immediately as secondary, but my attention has been called more than once to its presence as an evidence of syphilis by practitioners of good ability, whose care was proved by the fact that they observed the condition, so it may be well to emphasize the fact that *when an iritis develops in an eye which is inflamed in any other part, or contains a tumor, the probability is that it is* ***secondary,*** and does not depend on any general disease. Of course it is possible for an iritis to develop from some other cause in an eye which is otherwise inflamed, but this does not happen very often. When it is secondary to a conjunctivitis or keratitis we ascribe it commonly to the effect of toxines which have penetrated the capsule of the globe, diffused through the aqueous, and attacked the iris, but the inflammation may spread to the iris by contiguity from some other portion of the uvea, or from the sclera, or may be set up by the presence of a tumor, which seems to act in such a case like any other foreign body.

Traumatic Iritis, Cyclitis, and Iridocyclitis

An iritis may result from a wound of the iris made either by accident or design, or from an injury to the cornea or the lens, is plastic, as a rule, and may be of any degree of severity according to the nature of the injury. When the ciliary body is wounded the inflammation may be either plastic or serous at first, and be of any degree of severity between the two following extremes. If the wound is followed by intense pain in the head, vomiting, fever, œdema of the lids, chemosis of the conjunctiva which fails to conceal a very pronounced ciliary injection, tenderness of the eyeball, and by the formation of exudates behind the iris, in the pupil, in the vitreous, and in the ciliary body itself, a severe plastic iridocyclitis has started which will probably cause a complicated cataract and phthisis bulbi by cutting off the nutrition of the eye. In some of these cases the pupil will be found to be dilated because of the preponderance of the cyclitis over the iritis and the contraction of exudates in the ciliary body. If pus appears in the anterior chamber sepsis is present, and we are dealing with a purulent cyclitis which soon will develop into a panophthalmitis.

The other extreme is seen when a wound of the ciliary body seems to heal kindly, but the eye continues to maintain a little tenderness over the ciliary region and a tendency to be irritable, that is, to become injected, to lacrimate, and to show some photophobia on slight provocation. The danger of sympathetic ophthalmia is just as great in these cases as in the preceding, if not even greater because of the difficulty met with in trying to convince patients of the risk they are running. The danger of sympathetic ophthalmia is great in all cases of wound of the ciliary body, though occasionally we meet with a person who has carried such a wounded, irritable eye for many years without losing the other.

Iritis Met With in Acute Infectious Diseases

An iritis may occur in the course of any of these diseases, and is supposed commonly to be caused by metastasis of the characteristic microorganism. Typhoid bacilli have been found in the iris in typhoid fever, pneumococci in cases of pneumonia, and bacteria coli in those of enteritis, and sometimes it seems to be excited by the filariæ circulating in the blood of a person suffering from malarial

poisoning. In other diseases it may perhaps be caused by toxines, for no influenza bacilli have been found in any case of influenzal iritis. Such cases as these are rare, and are recognized easily.

Syphilitic Iritis

When a patient presents the typical symptoms of an iridocyclitis in one eye, that is, a swollen upper lid, an extensive, dark, ciliary injection, dustlike deposits on Descemet's membrane, a discolored iris with blurred details, and posterior synechiæ, our first thought is apt to be of syphilis, especially when he is young.

It may be possible for syphilis to excite a superficial iritis. Evidence to this effect can be drawn perhaps from the rare cases described by *Roemer,* in which a very early roseola, associated sometimes with a superficial papule, has been observed on the iris about six weeks after infection. Reddish spots appear on the ciliary zone, caused by congeries of blood vessels, possibly with minute, bright red nodules, and soon disappear spontaneously. Perhaps the reason why they are not seen oftener is that they cause no trouble and soon disappear. *Roemer* also says that *Krueckmann* has called attention to a condition in secondary syphilis in which whitish spots appear on the ciliary zone, especially about the crypts, in which the deep vessels look like white branches, with no preceding iritis. But clinical experience leads us to believe that such a superficial syphilitic iritis is exceptional.

In the majority of cases of **syphilitic iritis** we find the iris more or less thick and œdematous, with posterior synechiæ that are dentate, yet sometimes mixed with linear ones, and a hyphæma, or a jellylike exudate, often is present in the anterior chamber, but none of these conditions are diagnostic of syphilis. The only cases in which a diagnosis of syphilitic iritis can be made with any approach to certainty from the appearance of the iris are those of the papular form, which will be described presently. We must rely on the history and the presence of other clinical evidence of the disease. A positive Wassermann alone is not conclusive evidence as to the nature of the lesion, for other causes are competent to excite an iritis in a person infected with syphilis.

An iritis may be the ***first secondary symptom*** and appear about six weeks after the initial lesion of syphilis, but this is unusual. In such a case the presence of a chancre will suffice for the diagnosis,

but if this cannot be demonstrated we shall be obliged to await the onset of the other secondaries, which will appear in a few days. In the majority of cases iritis is one of the ***late secondary manifestations*** of the disease, and comes on between the third and the twelfth month after infection, when the presence of a characteristic cutaneous eruption, of mucous patches in the mouth, or of hard, painless, enlarged lymphatic glands, render the diagnosis easy. Sometimes it occurs during the second year, after the other secondaries have disappeared, and then we have to rely on the history, a positive Wassermann, and the exclusion of all other conditions that ordinarily excite this trouble. Iritis occurs in only a small percentage of the persons who acquire syphilis and is looked upon by some authorities as an indication that the disease is more than usually grave. This gravity seems to be increased when it assumes the papular form.

The best of treatment may not be able to prevent the outbreak of this inflammation, which has been known to appear after treatment had been discontinued because the patient was apparently well. In such cases other causes must be rigidly excluded. Only one eye is affected in the majority, but in a large minority the second eye is attacked after an interval. Relapses are common and may occur in each organ alternately. A thorough examination of the eye is likely to reveal an involvement of many other tissues. Sometimes a deep opacity can be seen in the cornea near its periphery, which resembles an interstitial keratitis, and then we shall probably find the underlying portion of the iris to be more swollen than the rest. The papilla often is hyperæmic, and sometimes is the seat of an optic neuritis. The retina may be inflamed primarily or secondarily, or we may see signs of choroiditis. The vision may be reduced to scarcely more than perception of light by the cloudy vitreous when the choroid is involved, and yet be regained almost perfectly under energetic antisyphilitic treatment.

The prognosis of the purely fibrinous form is pretty good under energetic treatment, though relapses cannot be guarded against, and the vision remains impaired more or less in a great many cases. Each relapse in every form of iritis increases the number of posterior synechiæ, and this increase may lead to exclusion and occlusion of the pupil, especially when the disease is neglected. The result of this may be crater pupil, atrophy of the iris, secondary glaucoma, complicated cataract, and phthisis bulbi.

The Papular Form of Syphilitic Iritis

When we see an iritis with little hyperæmic **nodes** in the region of the sphincter we may be practically certain that we are dealing with a syphilitic manifestation, but we must always confirm this suggestion by other evidence of the presence of this disease. The nodes often are called gummata, but a better name is **papules** because it is expressive of their nature, which differs from that of gummata. Their appearance when the attack of iritis occurs early in the secondary stage of syphilis differs considerably from that seen in late attacks, so we distinguish between early and late papules, though no sharp line of demarcation can be drawn between them as we meet with many transitional forms. **Early papules** are small, quite red, apt to be more numerous than the late, and are to be observed more frequently. They may form a red ring about the pupil, though they seldom exceed six in number. The nodular condition of this red ring serves to distinguish it from the uniform or finely striated redness of the region of the sphincter sometimes seen in an iritis secondary to a serpiginous ulcer of the cornea, or due to pneumococcal infections of the interior of the eye. When the œdema of the iris is very great, even large papules may be buried in it and manifest themselves only as an irregular swelling at the margin of the pupil. The less fluid there is in the exudate, the more prominent they become.

Late papules may be single, are usually few when multiple, are larger than the early ones, are grayish or yellowish because the hyperæmia is less, have smooth surfaces, and sometimes look as though they were rather dry, especially when œdema is absent. The later they are the larger and more solid they appear to be. Rarely they have been known to extend out beyond the minor circle into the ciliary zone, when we are apt to find several confined to one sector of the iris, the largest ones at the periphery, where they indicate a similar affection of the ciliary body, the smallest at the pupillary margin. This is the **group syphilide.** These late papules perhaps may form an intermediate stage toward the formation of gummata, for sometimes, even with the microscope, we are unable to determine whether one of them is or is not a gumma, and true gummata are said to have appeared in the iris during the secondary stage of malignant syphilis.

All of these papules originate in the stroma of the iris and may grow rapidly or slowly. The rapidity with which they may increase

in size is shown by the fact that a rapidly growing, cellular papule has been taken for a hemorrhagic infarction. A papule of this kind may rupture and produce a hyphæma in which a yellow zone perhaps may be seen.

The response to treatment is not always the same. Early papules become absorbed and may leave either no traces, or atrophic spots in the sphincter. The involution of a late one frequently begins with a central softening and the papule flattens as absorption proceeds, or it may rupture and evacuate its contents into the anterior chamber; in the latter case we may see the node replaced by a little red mass of granulation tissue. After these papules have disappeared they leave spots that mark not only the injury that has been inflicted on the stroma and the chromatophores, but also atrophy of the muscular tissue of the sphincter, and remain permanently as characteristic signs that the patient has suffered from this form of iritis.

The prognosis of the papular form is not as good as that of simple fibrinous syphilitic iritis. Histological research seems to show that the papules are far more common than they appear to be clinically, that in most cases they are too small to be visible, and that an increase in their size seems to indicate an increase in the seriousness of the condition. *Roemer* states that statistics have shown one half of the eyes thus diseased to become blind. Parasyphilitic diseases of the central nervous system, tabes and general paresis, have been observed to appear later in many cases.

Syphilitic papules may be distinguished from the rare vascular and other tumors of the iris by the violent iritis with which they are associated, as tumors of the same size seldom cause any irritation. They may be differentiated from tuberculous nodules by their preponderance in the pupillary zone, the reddish color of the early ones, the absence of a local reaction to tuberculin, the presence of a positive Wassermann, as well as of other clinical signs of syphilis, and their response to antiluetic treatment. The iritic nodules of leprosy are attended by other symptoms of that disease, are found in the peripheral portion of the iris as a rule, and usually the iritis is chronic. The nodules of ophthalmia nodosa are associated with the characteristic symptoms of this disease in the conjunctiva.

Gummatous Iritis

This variety of syphilitic iritis is met with very seldom. A single, large, yellow tumor may be seen to lie in the iris of one eye near its ciliary margin with the surface about it lying in folds, to be recognized at once as a tumor that has started in the ciliary body and has pushed its way through the iris. It may be a **gumma,** a **solitary tubercle,** or a **sarcoma,** and often the clinical differentiation is difficult. If the growth is attended by a severe iritis, other signs of tertiary syphilis, a positive Wassermann, and it is influenced by antisyphilitic treatment, the diagnosis of a gumma may be considered positive. If the Wassermann is negative, and a local reaction is obtained from a subcutaneous injection of tuberculin, it is a tubercle, but a tubercle is not excluded by a positive Wassermann; we may have to decide from the preponderance of the symptoms of tertiary syphilis and of tuberculosis, or depend on the effect of treatment for our diagnosis. A sarcoma usually is darker and more vascular than either of the others, grows slowly, and excites no inflammation until it is quite large. It is still more uncommon than a gumma or a tubercle, and is not affected by any medical treatment.

Probably a gumma of the iris starts in all cases from the ciliary body. Its appearance may or may not be preceded by a plastic iritis. It may break down and form a hypopyon, or an opacity may form in the corresponding place in the cornea, which later may be perforated by the growth. Descemet's membrane always is studded with deposits. Cataract and secondary glaucoma are frequent complications. In other cases a gumma of the ciliary body forms a bluish black protrusion of the sclera and perforates at that point. The prognosis is bad. The eye is lost in most cases, and when it is saved the vision is likely to be much impaired. Sometimes the involution of a gumma has been known to leave an aperture in the iris.

Tuberculosis of the Iris

Modern research has taught us that tuberculosis of the iris may appear in many atypical forms, so when the ætiology of an iritis is at all obscure, especially in a young person, we should test at once with tuberculin. *Calmette's* instillation of a solution into the conjunctival sac *should never be made,* for fear of disastrous results. *Von Pirquet's* test leaves us still in doubt as to the nature of the

lesion, though, if the test is made perfectly, a negative result is a pretty good sign that there is no tuberculosis in the organism. The subcutaneous injection is the best method, for if it should excite a local reaction in the eye, whether it was associated or not with a general one, we should be fairly safe in concluding the iritis to be tuberculous, even though it presented no characteristic clinical symptoms.

When we see a number of minute, grayish yellow **nodules,** which vary in size from that of a pinpoint to that of a pinhead, scattered irregularly over the surface of an inflamed iris, confined mainly to the ciliary zone, but with perhaps a few small ones in the pupillary, we have reason to feel confident that the inflammation is tuberculous. The iritis is parenchymatous, as is shown by the œdematous appearance of the iris and the dentate posterior synechiæ.

We suspect its nature to be tuberculous when we see a **slow, chronic iritis** in which the entire iris is swollen, but presenting irregular lumps scattered over it as though the œdema was not uniform, though no nodules can be seen. These cases are complicated frequently by a crater pupil, and we must investigate the other eye carefully to make sure that it has not received a wound in order to exclude sympathetic ophthalmia, which often presents a similar picture.

When a **single yellowish tumor** is seen at the ciliary margin of the iris we suspect it to be a solitary tubercle if the patient is young, if its surface is rough and if it is accompanied by much fibrinopurulent exudate, while if the patient is older and is syphilitic, it is more likely to be a gumma. The differentiation in doubtful cases, and from sarcoma, has been given under gummatous iritis.

Tuberculous iritis occurs chiefly in young people and rarely is a complication of pulmonary or joint tuberculosis. In the majority of cases it appears to be ***secondary to disease of the lymphatic glands,*** especially those of the mediastinum and mesentery. Attention recently has been called to a chronic form that is said to attack young girls, and also women at the period of the climacteric, who seem to be otherwise in perfect health.

Gonorrheal Iritis

When a patient, particularly if he is a young man, suffers from an iritis and a simultaneous attack of articular rheumatism, it is

well to investigate for gonorrhea. Iritis rarely occurs during the acute stage of this disease, but is a rather common accompaniment of gonorrheal rheumatism, which attacks the joints during the late stages, or after the discharge has ceased. It is apt to be of the superficial type, and we often see at first a grayish exudate, composed of coagulated fibrin, at the bottom of the anterior chamber, which is not diagnostic, but is very suggestive of this cause. *Dernehl* has reported some cases that seem to show that the only accompanying sign from which we can make the diagnosis may be a chronic prostatitis, and that repeated milkings of the prostate may be necessary to find Gram negative diplococci.

This form of iritis is not only obstinate, but relapses are apt to be very frequent. Both eyes commonly are affected, though it may be alternately and at intervals. Exclusion of the pupil is not so very rare, and the iris may become atrophic in old cases. Total posterior synechia is seen sometimes. The iris alone may be affected at first, then the disease may spread to the ciliary body and the vision be impaired not only by the deposits of fibrin, the posterior synechiæ, and the pigmented spots left on the anterior capsule by their rupture, but also by the formation of dustlike opacities in the vitreous. For some reason with which we are not acquainted the choroid seldom is attacked.

We suppose the origin of this form of iritis to be metastatic, and that the gonococci give rise to toxic substances which maintain the inflammation for a long time, although they themselves are unable to live long in this tissue.

Gouty Iritis

When an elderly patient who is subject to gout is seized suddenly with intense pain in both eyes during the night, simultaneously with a typical attack of pain in the affected joint or joints, the chances are that he has an attack of gouty iritis. In the majority of cases we find both eyes very red from a conjunctival congestion that may mask the episcleral, deposits on Descemet's membrane, some discoloration and obscuration of the iris, and a few small posterior synechiæ; but in other cases these symptoms are not so marked, only one eye may be affected at a time, its onset may not be coincident with acute trouble in the joints, and sometimes the diagnosis cannot be made until a subsequent attack of gout renders it clear.

Possibly we should include under this heading the iritis that is said to appear occasionally in the children of gouty parents, and is considered to have a rather bad prognosis.

Rheumatic Iritis

When a patient complains of photophobia and lacrimation of one eye, which presents a congestion of the conjunctiva, or perhaps a slight conjunctivitis, has a normal pupil and shows *no recognizable signs* of iritis, the most skillful diagnostician may make a mistake, and probably has done so many a time, for these may be the first symptoms of a superficial iritis that develops slowly. After a few days we see a ciliary injection, a few vessels on the surface of the iris, and a tendency on the part of the pupil to contract, or perhaps to dilate slightly. It is well for the patient if we have suspected the true nature of the disease and instilled a mydriatic, but we are apt to hesitate to do this because most of the patients are elderly and we fear to excite a glaucoma. Usually we wait until the details of the iris are blurred by a deposit of fibrin, and then we are likely to find the characteristic linear synechiæ of a superficial iritis. Sometimes the entire iris and pupil are covered by a layer of fibrin which is stripped off by the dilatation of the pupil.

This rather unusual onset of an iritis is described under the heading of rheumatic iritis because it is met with almost if not quite wholly in the class of cases that formerly were included under this name, but it can no longer be considered to indicate rheumatism as its cause. It is simply one of the ways in which a superficial iritis may develop. In most cases the inflammation sets in rapidly with pain and the distinctive symptoms of iritis already described.

It is not uncommon for us to obtain a history of acute or chronic articular rheumatism, or at least one of indefinite pains in the joints or muscles, particularly in elderly people, but we must not accept these as necessarily indicative of the ætiology. All infections or localized foci of suppuration must first be excluded, and the diagnosis of rheumatism be confirmed by the finding of an excess of uric acid in the urine, before it can be taken into account as a possible cause. Still after everything else has been excluded a small number of patients remain in whom we can detect no other cause than a uric acid diathesis, and these we may perhaps term rheumatic,

whether the patients have rheumatic pains or not. The following case may be of interest as illustrative of this type.

A lady over 60 years old, a grandmother, was seized suddenly with pain in one eye, which presented the typical symptoms of a superficial iritis when seen a few days later. She was well nourished, in excellent general health, and asserted positively that she had never had a sore throat, a toothache, any rheumatic pains or twinges, or any other ache or pain in her life, aside from those incident to childbearing, until this trouble in her eye. Investigation revealed no signs of syphilis, tuberculosis, or any other disease, and nothing to direct attention to any focal point of infection, but her urine was found to be overloaded with uric acid and urates. After the excess of uric acid had been reduced somewhat by antirheumatic treatment the inflammation subsided. The treatment was persevered in for a long time after the iritis was well, until the excess of uric acid had been largely done away with; no relapse has taken place at the end of more than three years, and during this time she has developed no trouble which might suggest that a focus of septic inflammation had been overlooked. The uric acid diathesis commonly is associated with rheumatic pains, even though it was not in this case, so it seems reasonable to conclude that the diagnosis of rheumatic iritis, based on the excess of uric acid in the urine, was correct.

Rheumatic iritis never is purulent, and is not likely to result in atrophy of the iris. A slight attack may last only a few days, but one of ordinary severity is apt to persist for a number of weeks. Relapses are common.

Iritis from Anæmia

When the patient has no disease to which the iritis can be attributed, and the urine shows no excess of uric acid, it may be well to take into account the condition of the blood. Anæmia seemed to be the cause in a young man who had a very obstinate iritis associated with a diabetes insipidus, but with no history or symptoms of any disease known to produce such an inflammation. Examination of the blood revealed nothing except a simple anæmia, that of the urine nothing aside from the polyuria. Various lines of treatment were tried with no effect until finally iron alone was prescribed, when the iritis and the diabetes insipidus progressed rapidly and with equal steps to recovery.

Purulent Iritis

The iritis that accompanies an ulcer of the cornea, which has not perforated, often is associated with a hypopyon, the pus of which is free from bacteria and ***absolutely sterile.*** This may be called an **aseptic purulent iritis.** It is induced by the toxines of the micro-organisms present in the ulcer, which have become diffused through the posterior layers of the cornea and the aqueous and have excited an exudation of leucocytes from the iris. An aseptic purulent iritis rarely is excited by a general disease, though sometimes a periodic iritis with hypopyon is met with in malaria, a similar iritis with hypopyon occasionally is excited by influenza, and sometimes one appears as a symptom of an existing diabetes. It is a good rule to examine the urine for sugar whenever we meet with an iritis with hypopyon that is not associated with a corneal ulcer, or with an inflammation of some other part of the eye, and does not occur in the course of an infectious disease. The pupil may be occluded for a time by an exudate in diabetes, but a peculiarity of this exudate is that, as a rule, it is soon absorbed. Cases have been reported in which iritis occurred in diabetes without any hypopyon and with the formation of posterior synechiæ, but they are of even rarer occurrence than the others.

Septic purulent iritis generally follows a wound, or the perforation of a corneal ulcer, through which pyogenic organisms have gained access to the iris. Rarely it is brought about by metastasis in septicæmia and pyæmia, as well as in various bacterial diseases. Pneumococci have been demonstrated in iritis accompanying pneumonia, typhoid bacilli in one with typhoid fever, bacteria coli in one due to enteric trouble. Sometimes in these diseases the iritis is superficial with simply a hypopyon to show its purulent nature, in others the tissue of the iris is permeated and given a yellowish or brassy color. The prognosis depends on the virulence of the micro-organisms and the power of resistance of the tissues; it is good in mild cases, not so good when the inflammation is severe but confined to the anterior segment of the globe, as the vision then is badly impaired quite often, and bad when it extends back into the posterior segment, as it is then apt to go on to panophthalmitis.

ATROPHY OF THE IRIS

When we see in an uninflamed eye an iris that has lost its luster and the details of its design, so that it looks dull and bleached, and we can see the vessels stand out in it like cords, or perhaps like reddish lines, we know that it is atrophic. The stroma may have disappeared to such a degree that we can see the pigment of the retinal layer, or this layer also may be so thin that we can obtain through it a reddish reflex from the fundus with the ophthalmoscope. Sometimes apertures are to be seen where the entire tissue has given way, and the margin of the pupil is apt to be frayed. Such an atrophy may affect the entire iris, or only a portion of it, and always is due to some lesion that impairs the nutrition. Partial atrophies are found mostly in the region of the sphincter about synechiæ, sometimes elsewhere, as when left by the involution of a tubercle or a gumma, or caused by an iridodialysis. A senile atrophy occasionally appears in old people as the result of arteriosclerosis with no preceding inflammation, but much more commonly an atrophic condition can be traced to a chronic iritis, to repeated attacks of acute iritis, or to glaucoma. Not infrequently atrophy is caused by the constant stretching incident upon an adherent leucoma. In rare cases it is associated with a yellowish color, siderosis, and is then indicative of the presence of a piece of iron or steel in the eye.

TUMORS OF THE IRIS

A new growth presents itself as a rounded swelling that projects from the surface of the iris into the anterior chamber. If it is quite **small** we observe whether it is accompanied by iritis or not, for if it is we probably have to deal with a papular or a tubercular iritis, possibly with the lymphomata met with in leucocythæmia or pseudo-leucocythæmia, or the nodules of leprosy. The differentiation of the first two has been given already, lymphomata are recognized through the symptoms of the fundamental disease and an examination of the blood, leprous nodules by the presence of other symptoms of leprosy.

When no iritis is present we notice the color and the general appearance of the growth. If it is almost transparent it is a small serous cyst, which may be congenital, but in most cases has resulted

from a wound of the eye; when it has the luster of mother of pearl it is a pearl cyst, which always is traumatic in its origin and is usually due to the implantation of an eyelash into the iris; when it is grayish or yellowish we have to think of a possible congenital dermoid, and of the very rare entozoal cysts produced by filariæ and cysticerci. The last may be diagnosed if we can see within the cyst a whitish body that changes its shape from time to time. If the little tumor is dark it may be a congenital pigmented nævus, or a commencing sarcoma; the differentiation between these can be made only after prolonged observation, as the nævus will not increase in size, though it may become darker, while the sarcoma will grow slowly.

A **large tumor** almost always excites iritis, is accompanied by pain, causes an opacity of the cornea wherever it comes in contact with its posterior surface, and produces secondary glaucoma mechanically. A cyst is recognized through its more or less transparent walls, and its transparent or translucent contents. It is very exceptional for the wall to rupture and the cyst to undergo involution. A solid tumor may be a gumma, a tubercle, or some other neoplasm, usually a sarcoma. In many cases the differentiation is so difficult that it cannot be made until the tumor has been excised by an iridectomy and submitted to a microscopical examination, but sometimes it can be made fairly well by the positive exclusion of both gumma and tubercle, or through a history that shows the tumor to have grown slowly, and to have been present for a long time before it caused any inflammation, for the onset of both of the former is coincident with inflammatory symptoms.

THE SYMPTOMATOLOGY OF THE PUPILS

Mydriasis

When one pupil is widely dilated we inquire concerning a possible **contusion,** for a wide dilatation of the pupil may persist long after all other signs of a blow have passed away. We may be able to see a minute notching of its margin when the sphincter has been lacerated, particularly in fresh cases, but the tear rarely can be seen plainly, though in very exceptional cases it may extend beyond the sphincter into the ciliary zone. We must also consider the possibility that a solution of such a **drug** as atropine, homatropine,

daturine, duboiscine, hyoscyamine, or hyoscine has been instilled into the conjunctival sac. In both of these conditions there is no reaction of the pupil to light or to convergence, because the sphincter is paralyzed. If the anterior chamber is shallow we test the tension of the eyeball and look for other signs of **glaucoma.** If other muscles of the eye are paretic we look for the cause in a lesion somewhere along the course of the motoroculi, and if we can find no such lesion we search for a source of irritation to the **sympathetic nerve** on the same side.

If both pupils are dilated we have to consider the same causes, traumatism, the use of a mydriatic, and glaucoma, and when these are excluded we have to take into account whether the patient is suffering from pain, or is agitated by some emotion which causes a **reflex mydriasis.** In a child we must remember that mydriasis may be caused by the irritation produced by **intestinal worms.** If other muscles of the eyes are paretic we search for **intracranial trouble** so situated as to affect both motoroculi. If the patient is insane we think of **melancholia** and **mania,** if he has convulsions, of **epilepsy.** If he is very sick he may be suffering from **ptomaine poisoning,** or from **uræmia.** We may be guided to a tumor or an inflammation of the **spinal cord** by symptoms referable to these diseases, but in most of them the pupillary symptoms are unreliable. The **sympathetic nerve** may be irritated by the presence of cervical or mediastinal tumors that do not press it sufficiently to cause paralysis, or by the carbon dioxide in the blood during asphyxiation. Finally when a person faints the pupil is caused to dilate by an **ischæmia** of the iris.

MYOSIS

An abnormal contraction of the pupil may be due to a local cause, or to a general one; in the latter case it is known as spastic when it is caused by an irritation of the motoroculi, and as paralytic when it is due to a paralysis of the sympathetic. The differentiation between a spastic and a paralytic myosis can be made in most cases by the instillation of a drop of cocaine solution, as this usually fails to act in the former, but dilates the pupil in the latter.

We investigate first for **local causes,** such as a foreign body on the cornea or conjunctiva, a hyperæmia of the iris excited by an inflammation of a neighboring structure, or an iritis. After these

have been excluded we test the tension of the eye, for if this is subnormal it may account for the condition of the pupil. We must also exclude the possible instillation of a solution of eserine, pilocarpine, or arecoline. The myosis is unilateral in most of these cases, but a bilateral myosis may be more pronounced in one eye than in the other. When the patient is unconscious and both pupils are contracted we take into account the degree of coma, for if this is profound we have reason to fear **poisoning** from opium or morphine; if it is moderate the condition may be due to poisoning with alcohol, mushrooms, uræmia, or any other form of intoxication in which the action at first, or in moderate quantities, is irritant to the nervous system, though its effect may be paretic later on or in larger doses, when the myosis is replaced by mydriasis. A familiar example illustrative of the effects of these toxic substances on the pupil is seen in the general anæsthesia produced by ether and chloroform; during the stage of excitement the pupils are large and react to light, after the patient is fully under the influence of the drug they become small and do not react, and when the anæsthesia is so deep that the patient is in danger the myosis is replaced by mydriasis, the pupils remaining immobile. The pupils of a drunken man may be either contracted or dilated, and as both myosis and mydriasis may be caused by intracranial lesions it is very hard sometimes to determine whether a patient picked up unconscious in the street is suffering from alcoholism or intracranial trouble. The odor of the breath and the stertorous respiration alone are not to be relied upon, for many persons who do not drink habitually take a glass of liquor when they feel badly, and stertorous breathing is common to many **intracranial troubles.** If the pupils are contracted we should think of a traumatic or spontaneous apoplexy, especially of a hemorrhage into the pons, of an epileptic fit, of a tumor or abscess near the corpora quadrigemina or along the course of the motoroculi, and of poisoning.

If the patient is conscious we have to bear in mind the same cerebral lesions, which may not be of sufficient degree to cause unconsciousness, and to think of chronic opium or morphine poisoning, as well as of such diseases of the spinal cord as tabes dorsalis and general paresis. When myosis is associated with optic atrophy we have good reason to feel pretty sure of the presence of one of the last mentioned diseases, for in other forms of optic atrophy the pupil ordinarily is dilated. The suspicion that either tabes or gen-

eral paresis is present in such a case is strengthened if the myosis resists both cocaine and atropine. Myosis and blindness are not enough to form a basis for this diagnosis, as such a combination is met with sometimes in hysteria, but the optic nerve is not atrophic in such cases.

When no symptoms indicative of any such lesions can be found we should search for something that might cause a **paralysis of the sympathetic nerve,** such as a wound in the neck, an operation like excision of the superior cervical ganglion, or the presence of a tumor of any nature in the neck or thorax. After all of these pathological conditions have been excluded we may have to be content to ascribe the myosis to a ciliary spasm, if the eyes have been engaged in prolonged and hard near work, or to hysteria if they have not.

ANISOCORIA

When the two pupils are not of the same size we must not jump hurriedly to the conclusion that the patient is suffering from some disease of the central nervous system. It is quite possible that he may be suffering in this manner, but he may have a disease in one eye that makes its pupil larger or smaller than that of the other, or he may have some disease that induces the inequality through its effect on the sympathetic nerve, or the anisocoria may be a physiological condition. After excluding a disease of the eye itself we ***test the reactions of the pupils;*** if these are normal we have excluded an affection of the central nervous system, if they are not a lesion is situated there. If they are normal the next question is whether the patient is suffering from some disease of the lungs, stomach, kidneys, or other organs, that might affect the sympathetic. The majority of patients with such troubles are quite sick and the connection can be traced more or less easily, but anisocoria sometimes appears as an **early symptom of tuberculosis,** especially in children, in whom this disease is apt to appear first in the lymphatic glands, so anisocoria in a child should lead us to investigate the condition of his bronchial glands on the same side with the larger pupil. A tumor or an aneurysm in the thorax may produce the same symptom in the same way, by pressure on the sympathetic. If no evidence of any local or general disease can be found, and the pupillary reactions are normal, we feel sure that the anisocoria is physiological. It may be caused temporarily when a bright light

shines into one eye, but not into the other, or when the two retinæ are in unlike conditions of adaptation, but it is so slight as to be scarcely noticeable and soon passes away under the influence of an even illumination. Inequality of the reaction to the closure of the lids is said to have been the cause of a temporary anisocoria on very rare occasions. These being excluded the condition is congenital and of no importance. Sometimes we find the refraction widely different in the two eyes, sometimes one eye is blind or amblyopic, but in many cases nothing can be found to account for its presence.

When one or both pupils have the light reflex impaired in the absence of local disease, there is trouble in the **central nervous system.** If one pupil reacts properly while the other does not, we know that there is a unilateral lesion of the centripetal light reflex tract, or of the centrifugal tract for contraction of the pupil, but if the reflex of both is impaired the lesion is bilateral and affects the two sides unequally. Hence anisocoria is met with in the same diseases that cause reflex immobility of the pupil, and is useful as a symptom mainly in calling attention to the condition, and in helping to locate the lesion.

HIPPUS

Sometimes the governing power that regulates the size of the pupil to the amount of light that enters the eye seems to be at fault, so that the reflex contraction is too great and is followed immediately by an equally too great dilatation. In such a case the pupils constantly contract and dilate although there is no change in the light or in other conditions to account for such actions. This is called hippus. It may occur alone or with nystagmus, and it ceases during sleep. Perhaps it is met with most often in cases of multiple sclerosis, but it occurs also in cerebrospinal, purulent, and tuberculous meningitis, epilepsy, chorea, cerebral syphilis, and is said to appear sometimes during recovery from an oculomotor paralysis. The exact location of the lesion which produces this symptom is unknown.

Once in a great while we may see a case in which one pupil alone acts in this manner. Such a hippus usually is associated with a paresis of the motoroculi which was either congenital or was developed early in life, when the rise and fall of the upper lid may be synchronous with the contraction and dilatation of the pupil.

ALTERNATING MYDRIASIS

The contractions and dilatations of the pupils may alternate without apparent cause. Such a condition has been observed in persons who apparently were healthy, but *Frenkel,* who has studied the matter considerably, doubts if it ever occurs in the absence of disease, and is inclined to ascribe it to a spasmodic action on the cervical sympathetic, which may be either direct or reflex. We think first of **general paresis,** as it has been observed most often in this disease, and then of tabes, unless the patient is suffering from pneumonia, pleurisy, peritonitis, or some similar disorder. More rarely it has been seen in chronic cerebral myelitis and hydrocephalus, and one case is on record in which it occurred in infantile paralysis of cerebral origin. Cases in which it has been ascribed to insular sclerosis, exophthalmic goiter, neurasthenia, and hysteria, are open to doubt, in *Frenkel's* opinion. Recently I saw a marked case in a boy who had been brought into the hospital unconscious as the result of an accident in which he had been struck on the head. Symptoms of increased intracranial pressure became so severe that a double decompression operation had to be performed and some blood was evacuated, but the site of the hemorrhage could not be ascertained. In this case the light reflex of the pupils was thought to be good at first because they were observed to contract as light was thrown upon them, but after a few moments it was noticed that they would dilate alternately and then contract without any reference to whether the light was shining into them or not, so it was considered doubtful if they actually responded to the light. This alternate dilatation and contraction of the pupils from no apparent cause cannot be mistaken for anything else.

HEMIANOPIC PUPILLARY REACTION

In some cases of hemianopsia we may be able to obtain information as to the site of the lesion producing it by ascertaining whether the pupil does or does not react when the affected side of the retina is irradiated, but the test is so delicate and difficult to make that little can be hoped for from it except in very experienced hands, and even then it often is unsatisfactory. The patient is placed in a dark room and the region of the pupil is illuminated by a light placed at his side, so weak as to be only just sufficient to enable us to see

any movement that may be made. A beam of stronger light is then thrown obliquely into the eye so as to fall on the hemianopic side of the retina. It is desirable to have this beam fall as near the center as practicable, because the pupillomotor power of the retina seems to decrease toward the periphery, whether there is a pupillomotor area or not. The beam must be small because light is reflected and diffused very readily within the eye to all parts of the retina. The beam is then turned to fall upon the opposite side of the retina, about equally distant from the center. If no pupillary reaction takes place when the light falls upon the hemianopic side, but appears when it is thrown on the opposite, the lesion is in front of the external geniculate body, but if the pupil contracts when the light is thrown on either side, it is farther back. The inherent difficulties of this test are that when we fail to elicit a response we have to question whether the light was strong enough to stimulate the part of the retina upon which it fell, and when the response is prompt, whether it has not been caused by diffusion of the light. We may decide in the former case by being careful to throw the light upon a corresponding point of the retina on the opposite side, but the decision in the second must depend on experience and judgment.

SLOWNESS OF REACTION OF THE PUPIL TO LIGHT

The reaction of the pupil to light may be slow, or diminished to any degree up to immobility. Such a slow and imperfect reaction is one of the symptoms of hyperæmia of the iris, iritis, and glaucoma, when it is accompanied by other symptoms of these conditions, and frequently it is seen in diseases that affect the macula, like a central choroiditis. As we would naturally expect, it is present in varying degrees during the recovery of the sphincter from paralysis due to any cause, and it may be a forerunner of an immobility of the pupil caused by a lesion of the central nervous system.

AMAUROTIC IMMOBILITY OF THE PUPIL

An eye which is totally blind has lost its direct pupillary reaction to light, but retains its consensual reaction. This furnishes us a test by which we can ascertain whether an eye really is blind or not; if the pupil reacts directly to light the eye almost certainly is not

blind, while if the pupil fails to respond directly, but reacts consensually, the eye cannot perceive light. At the same time the pupil will react perfectly to convergence. The pupil of the blind eye is rather larger than that of the other in reduced light, but not noticeably so in ordinary illumination. When both eyes are blind there is neither direct nor consensual reaction and, as a rule, the pupils are dilated, though in some diseases of the central nervous system they may be contracted. It may still be possible to secure a contraction of the pupils through the attention reflex, or through the reflex to the closure of the lids, or to obtain a dilatation through a sensory or psychic stimulus.

ABSOLUTE IMMOBILITY OF THE PUPIL

When one or both pupils are dilated and refuse to react to either light or convergence they are said to be absolutely immobile. Perhaps the most common example of this is the condition produced by the instillation of atropine, or of any other **cycloplegic,** but it occurs not infrequently as the result of a **contusion** of the eye. It may accompany **ptomaine poisoning, lead poisoning,** and **cerebral syphilis.** This last disease is the one we think of first when only one eye is affected, and the instillation of a cycloplegic can be ruled out, when we may or may not find the ciliary muscle paralyzed in spite of the immobility of the pupil. When both pupils are absolutely immobile and the ciliary muscle is paralyzed the condition is so very typical of a **postdiphtheritic paralysis** that it often is accepted as evidence that a preceding inflammation of a mucous membrane was diphtheritic, though it has been said to follow influenza in rare cases. It should be admitted that the accuracy of the last claim is not considered proven. A lesion situated in the sphincter, short ciliary nerves, ciliary ganglion or its motor roots, the trunk of the motoroculi, or the ganglion cells of the nucleus of the sphincter, may give rise to an absolute immobility of the pupil; if it is in front of the ciliary ganglion eserine will not contract the pupil as much as usual, but if it is located farther back the drug will produce its ordinary effect. It is also possible to produce an absolute immobility of the pupil by inhibition of the sphincter through the cortex of the brain, and we meet with it occasionally in **general paresis,** less often in tabes.

REFLEX IMMOBILITY OF THE PUPIL

When a pupil does not react either directly or consensually to light, but responds promptly to convergence it is said to be in a state of reflex immobility, and is called the **Argyll-Robertson pupil.** Myosis or mydriasis may be present or the pupil may be of its normal size, but it fails to dilate in response to sensory and psychic stimuli, as well as to react to light. In the great majority of the cases an Argyll-Robertson pupil is indicative of either **tabes,** or of general paresis, usually the former. Some writers mark it as an isolated symptom in syphilis, but it is less common in cerebral syphilis than absolute immobility of the pupil, which is met with less often in general paresis, and still less often in tabes. Reflex immobility is said to have occurred in cerebrospinal diseases which did not appear to be of syphilitic origin, including a few cases of multiple sclerosis, syringomyelia, and hypertrophic interstitial neuritis, and it is also said to occur in insanity.

The unchanging pupils give a peculiar, fixed expression to the eyes. Often we find them rather irregular, and sometimes the iris becomes atrophic, its surface flattened, and its luster diminished. This atrophy may involve only a single sector of the iris, and then, according to *Dupuy-Dutemps,* it has a distinct influence on the shape of the pupil. As the atrophy advances the reaction to convergence becomes progressively weaker.

Quite a number of theories have been advanced to explain the mechanism of reflex and of absolute immobility of the pupils, but we do not know with certainty that any of them are correct.

When we come to sum up what we can learn from observation of the various disturbances of the pupil which have now been described, we are compelled to admit that the particular nature of the disturbance in the pupil is, on the whole, a very unreliable guide to a diagnosis when taken by itself. In acute alcoholism the pupils often are contracted, often dilated, sometimes normal in size; their reactions may be increased, decreased, or not affected. In chronic alcoholism we often find them rather small and their reactions slow, but this is not always the case. The same is true in most cases of poisoning; the quantity of poison taken, the nature of its action, the susceptibility of the patient, and the stage of its action at the time we see the patient, all modify the effect on the pupils, which may be produced directly, or indirectly through the faulty

oxygenation of the blood. In either case the effect may be irritant at first and later paralytic, so what we find depends on many causes. The behavior of the pupil is variable likewise in meningitis and the majority not only of intracranial troubles, but also of psychoneuroses and mental diseases. Nevertheless, the fact that the pupil is deranged is a valuable asset in diagnostic work. In dealing with disease of the eye itself, the nature of the pupillary disturbance is of great help, but for diseases outside of the eye there are only three of the central nervous system which may be said to have characteristic pupillary symptoms, tabes, general paresis, and cerebral syphilis, to which we can add the absolute immobility of the pupils in postdiphtheritic paralysis of the accommodation, and the chronic myosis caused by the habitual use of opium or morphine.

CHAPTER XI

The Lens

Our attention may be called to the lens by a white spot in the eye, which is quite apt to have been noticed by the patient himself and associated by him with an impairment of his vision, or we may be led to investigate its condition by a history of visual trouble that followed a traumatism; but, unless the changes have produced a whitish opacity which is apparent to anyone, an affection of this organ is not unlikely to escape observation, because the subjective symptoms induced by it are extremely diverse, and, as a rule, not characteristic. For this reason we should make a systematic examination of the lens whenever a patient presents any visual or other anomaly of the eye that is not accounted for otherwise, or has a vague feeling of discomfort, or easily induced weariness. Some of the abnormal conditions met with are congenital, others are acquired. Some demand immediate operative intervention, others are not urgent, but are curable by operation, while still others had better be left alone. The lens may be displaced from its normal position in the patellar fossa, its transparency may be impaired, it may be irregular in form, and it may be swollen or shrunken.

DISLOCATION OF THE LENS

When the lens is displaced from its normal position it is said to be dislocated. This condition may be congenital, when it is called ectopia, or appear during later life, either spontaneously or as the result of traumatism, when we refer to it as luxation if the lens has been dislocated completely, or as subluxation if a part of it remains in the patellar fossa. Possibly ectopia should be held to include spontaneous dislocations which appear in children after birth, and are dependent on a congenital fault.

Luxation and Subluxation

If we find the anterior chamber of an eye deeper on one side than on the other and the iris in the deeper portion to be tremulous,

we know that the iris at this place does not receive its normal support from the lens. We then turn our attention to the pupil, perhaps after dilating it with a mydriatic, and see if we can find it divided by a sharply defined curved line which separates a very dark gray portion within its concavity from a deep black one on its outer side. This line is bright and shining when it is viewed by oblique illumination, black when seen with the ophthalmoscope, and is the edge of the dislocated lens. The refraction is widely different in the two parts of the pupil, highly hypermetropic in the deep black outer portion, because the eye is aphakic at this place, while it is very likely to be myopic and irregularly astigmatic in the other, slightly grayish portion, because this part is occupied by a lens which has been allowed to assume as nearly a spherical form as its age will permit by the rupture of the zonule. This is a **subluxation** of the lens. The lens has been moved from the center of the fossa patellaris, decentered from the rest of the refractive media, and perhaps tilted so that one edge looks forward more or less. If the subluxation is enough to permit the edge of the lens to divide the undilated pupil, the patient probably has monocular diplopia; otherwise his vision is simply blurred, provided that no other trouble is present.

In another case we find the anterior chamber evenly deep, the entire iris tremulous, the pupil a deep black, the refraction highly hypermetropic, and then we know that the lens is wholly absent from the fossa patellaris. It may have been removed or extruded from the eye, but if it remains anywhere within the organ it is said to have been **luxated.**

Both subluxation and luxation may be either spontaneous, or the result of traumatism. In the former case we need to look for a disease that would cause a degeneration of the zonule, such as a high degree of myopia, or a choroiditis, but we must remember that after the zonule has been weakened by disease the actual dislocation often is produced by a jolt or jerk of the head caused perhaps by a sneeze, or by a concussion that did not affect the eye directly. This is a point of medicolegal importance, and it is only when such an accident can be ruled out that a dislocation of the lens should be called spontaneous.

The lens may be luxated backward into the vitreous, where it may do little damage, or may act as a foreign body and set up a destructive inflammation, or it may be luxated forward. If the

anterior chamber is unevenly deep, while the pupil is large, oval, and immobile, it is practically certain that the lens lies in the aperture formed by the pupil with one part projecting into the anterior chamber, and it is likely that we can see the shining edge of this part by oblique illumination. In another case the anterior chamber is very deep everywhere and we can see by oblique illumination that the aqueous has been largely replaced by something that resembles a drop of oil with a bright margin; then we know that the whole lens has been luxated into the anterior chamber. In both of these cases the vision of the eye is much impaired, the patient complains of severe pain that radiates through the side of the head, and a ciliary injection soon appears. These symptoms increase steadily in severity, the tension of the eye rises, and secondary glaucoma rapidly supervenes. Ordinarily the lens loses its transparency quickly and then we can see it more easily. By its presence in the anterior chamber it will destroy the eye very soon, and its removal is a matter of urgency.

The lens is luxated forward sometimes as the result of a sudden perforation of a corneal ulcer, and can even be extruded from the eye when the opening is large enough, as happens occasionally in ophthalmia neonatorum. It can also be extruded through a rent in the capsule of the eye caused by a contusion.

Sometimes, though very seldom, a luxated lens that falls back into the vitreous when the patient lies upon his back may be made to pass through the pupil into the anterior chamber when he shakes his head while he holds it bent forward. Such a lens must be shrunken, and the difficulty of extraction is lessened if we can get it into the anterior chamber and secure it there by contracting the pupil with eserine.

A luxated or subluxated lens may remain transparent, but is very apt to become opaque after a short or long interval. It is at all times a source of danger to the eye, yet its extraction is not an operation to be undertaken lightly, for in many cases it is one of the most difficult in ophthalmic surgery to bring to a successful conclusion.

Ectopia

A traumatic or spontaneous dislocation of the lens rarely is bilateral, and when it is present in both eyes the displacement is very unlikely to be symmetrical, but these conditions are reversed

when we have to deal with what is known as congenital ectopia. Frequently this is detected during early life in children who belong to families in which it is hereditary, or when it occurs in association with corectopia, irideremia, or nystagmus, but often it remains unsuspected until the child goes to school and is found to have poor vision. Then he may be thought to be nearsighted and a series of experiences with opticians and optometrists may precede his coming under our observation. The condition may not be apparent at first unless we notice that the lower part of each anterior chamber is deeper than the upper, and that the lower part of the iris is tremulous. The child may seem to be simply amblyopic, but when we dilate the pupils for the purpose of retinoscopy our attention is attracted by a dark curved line in the lower part of each, and we find the lenses subluxated, usually symmetrically upward, perhaps with an inclination outward or inward from the median line of the eye. We are likely to recognize the condition more quickly if the patient has corectopia, which usually is upward with the lenses, irideremia, or coloboma of the iris, or if he complains of polyopia. In the last case we shall probably find the edge of the lens to be visible in the undilated pupil. It is only in rare cases that the lenses are displaced downward, inward, or outward, and still more rarely is the displacement asymmetrical. The lenses seldom if ever are tilted.

Ectopia is hereditary, and the particular type seems to be transmitted from one generation to another, but we occasionally meet with sporadic cases.

The prognosis is uncertain. The lenses may remain clear and not change position for many years, perhaps not for life, but they are luxated more readily and by slighter traumatisms than those which occupy their normal positions. Many of them exhibit a tendency to move upward slowly, to shrink, and to become cataractous. Sometimes it is difficult to say that an ectopia of the lens was actually congenital, for a perfectly typical case may develop during childhood. In an irideremic girl five years old I found the lenses clear and in normal position, the eyes rather amblyopic, but no refractive error except a simple myopic astigmatism. Three years later there was considerable myopia, the lenses were slightly ectopic upward and exhibited striæ of opacity, a condition that differed in no way from a congenital ectopia with commencing cataracts.

COLOBOMA OF THE LENS

A coloboma is a notch or concavity in the lower margin of the lens, rarely elsewhere, and in about half of the cases in which it is found it is associated with a coloboma of the iris, of the choroid, or of both. A very large one may give rise to irregular astigmatism, but a small one causes no visual trouble and is discovered accidentally, as a rule. Sometimes a narrow strip seems to have been cut away in so wide a sweep that the lower margin appears in the pupil, when it is dilated, as a nearly horizontal line, and this may readily suggest ectopia, or subluxation upward. The differentiation can be made usually from the absence of visual trouble, from the lack of a history or of any symptoms pointing to traumatism, from the lack of a disease likely to affect the zonule, and from its presence in one eye only. The cause of such a flattening of the margin of the lens is problematical, though various theories have been propounded.

LENTICONUS

Occasionally a child is brought to be fitted with glasses because of nearsightedness, and we find that while the refraction near the center of the pupil is highly myopic, that in the periphery is much less so, perhaps emmetropic, or hypermetropic. An adult may inform us that he has become nearsighted recently, and show a similar condition of his refraction; if he is quite elderly he may be jubilant over his "second sight," as he is able to read without glasses again, and he may not be aware that he cannot see well at a distance, for old people often are oblivious to such a fact. We first examine the cornea in each case to exclude keratoconus, and if its curvature is normal we know that the fault is in the lens.

In the child we are likely to find that either the posterior or the anterior surface of the lens projects in the form of a cone, less often in that of a sphere, a congenital fault of development which is not apt to change and is called **posterior** or **anterior lenticonus.** The posterior is by far the more common, and usually is associated with an opacity at the posterior pole, sometimes with opacities elsewhere in the lens. In the adult it is more likely that a change has taken place which has produced an abnormal difference between

the indices of the cortex and of the nucleus, a condition called **false lenticonus** as the form of the lens is not changed. In the majority of cases false lenticonus is a forerunner of cataract, but occasionally an old man retains his second sight for a long time.

The differentiation between these forms of lenticonus can be made by means of Purkinje's images. If the posterior one is very small at a certain place and becomes elongated when the light is moved a little to one side, the condition is one of posterior lenticonus. If the anterior image behaves in the same way the condition is one of anterior lenticonus, and we may be able to make out the conical shape of the anterior surface of the lens by oblique illumination. If both images are of their normal sizes but the posterior one is unusually bright, we know that the patient has a false lenticonus.

CATARACT

Any opacity of the crystalline lens, of its capsule, or of both, is known as a cataract. Such an opacity may appear at any time during life from before birth to extreme old age, may be caused by traumatism, may be symptomatic of disease, or may develop spontaneously as the result of processes with which we are at best imperfectly acquainted. The consistency of the lens varies with the age of the patient; it is soft in childhood and develops in early manhood a small hard nucleus which increases in size until it renders the lens almost wholly hard in old age; so a cataract may be accordingly soft, without or with a hard nucleus, or hard. Sometimes a cataractous lens degenerates and its fibers become liquefied, while lime salts may be deposited in it or its capsule, so as to form a **fluid,** or a **calcareous** cataract, and occasionally the fluid becomes absorbed until little is left of the lens except its capsules, which form a sort of membrane. The color in the great majority of cases is white or gray, when the lens is visible as a white spot in the pupil posterior to the plane of the iris, but exceptionally it is amber or brown, as in black cataract. The opacity may occupy the whole lens, or only a portion of it, and in the latter case may have many situations, as is seen in the different varieties of congenital cataract. When it is secondary to, or is complicated by, an intraocular inflammation, the cataract is said to be **complicated,** otherwise it is simple. A cataract that develops in an elderly person from an unknown cause is called **senile,**

and may be observed in its incipient, immature, mature, or hypermature stages, according to the period of its development.

Congenital Cataract

We are unable to differentiate a cataract that was present at birth from one that developed during the first few years of life, unless informed by the history, so we are accustomed to call all which appear during childhood congenital when they cannot be explained as the results of traumatism, or of disease. The patient is a child, as a rule, but sometimes is an adult who states that he has had the trouble ever since he can remember. Ordinarily the diagnosis is not difficult, whatever may be the age of the patient. Congenital cataract affects both eyes almost invariably, and most of its forms are so characteristic that they are recognized as such at once when they are bilateral, unless there is evidence that they were produced by a known cause. A cataract in one eye alone is so very likely to have been acquired that it can be called congenital only when it is positively known to have been present at or shortly after birth, to have not changed for a long time, and that the eye has not suffered from traumatism or disease.

Zonular or Lamellar Cataract

The most common form of congenital cataract is the zonular or lamellar. We see by oblique illumination a round gray opacity, the center of which appears to be less dense than the rest, surrounded by a transparent zone, behind the plane of the pupil in both eyes. After we have dilated the pupils with a mydriatic we see with the ophthalmoscope that this opacity forms a disk lying in transparent lens tissue, that its center really is less opaque than its margin, and that little dentations extend out from the latter into the clear tissue. If we use a high magnifying power we can see that the opacity consists of innumerable minute points and larger grayish clouds, while part of the dentations seem to ride on the margin, having two legs, one coming from the anterior, the other from the posterior layer. Some of these riders are separated from the opacity and lie in the clear tissue, where they indicate the commencement of a new layer. Occasionally one or two opaque zones may be seen about the principal opacity.

The size of the opacity varies; it may be so small as to impair

the vision only a little, and a very few have been reported as rudimentary, but, as a rule, it is large enough to nearly or quite fill the pupil. It is developed uniformly in both eyes almost always, and the only cases in which a question arises as to its congenital origin are the rare ones in which the cataracts are unequal, or are not present in both eyes; then we have to make a careful inquiry to ascertain whether the eye has not suffered from ophthalmia neonatorum, the perforation of a corneal ulcer, iritis, or traumatism.

The degree of vision depends not only on the size of the opacity, but also on the thinness of its center, which sometimes is almost diaphanous. We must ascertain how much it can be improved by glasses, when the patient is old enough to be tested, and whether it can be helped by dilatation of the pupil, before we decide concerning whether any treatment should be given, as it is exceptional for any changes to occur in the extent or the density of the opacity after this time, though probably changes do take place during the very early years of life.

Occasionally we need to differentiate a zonular from a membranous, or a siliquose cataract, and we do this by observing the conditions of the anterior chamber, iris, and pupil. In an uncomplicated case of zonular cataract the depth of the anterior chamber is normal, the iris is not tremulous, the pupil is round, free, and active, while in both of the others the chamber is deep, the iris is tremulous, and often is adherent to a secondary membranous cataract so that the pupil is irregular and hampered in its movements.

Total Cataract

Another rather common variety of congenital cataract is the total, in which both lenses are completely opaque. They are uniformly white and the diagnosis is easy. It is sometimes difficult to tell whether they are advanced zonular cataracts, or were caused by vascular trouble in the embryo, perhaps by a perivasculitis of the vascular capsule; but in most cases this is not a matter of vital importance.

Central Cataract

Occasionally we find a round, white dot in the center of the nucleus, which affects vision very little and is not accustomed to

progress. Such a central cataract may be present in both eyes, perhaps in association with other opacities, or we may find it in one eye and some other form of congenital cataract in the other. This form is always congenital, is hereditary, and bears no resemblance to the nuclear variety of senile cataract, which appears in advanced life as a cloudiness of the nucleus.

Fusiform or Spindle Cataract

Once in a great while we see lenses in which a tubular or spindle-shaped opacity extends from the anterior to the posterior pole of the lens, or from the former to the nucleus. Such an opacity is always congenital, whether it is accompanied by others or not, and is not to be mistaken for anything else.

Posterior Polar Cataract

Sometimes we are able to detect an opacity, frequently no larger than a pinhead, at the posterior pole of the lens. The only subjective symptom that can be considered at all characteristic is a speck to the outer side of the patient's line of vision which is constant, but continually eludes his attempts to fix upon it. What we see is a gray spot situated deeply in the lens, which we ascertain to be behind the nodal point through its parallactic displacement in the direction opposite to that in which the eye is moved, but it is not always easy to demonstrate whether it is due to a change in the posterior capsule, or in the posterior fibers of the lens. If it happens to be associated with some other form of congenital cataract, posterior lenticonus, or remains of the hyaloid artery, we feel pretty sure that it is congenital, but if none of these things are present we examine the fundus in search of pigmentary degeneration of the retina, or choroiditis, both of which may induce the defect through the formation of an opaque spot at the posterior pole of the lens, probably as the result of a disorganization of the vitreous. This spot may show some radiating lines when it is caused by pigmentary degeneration of the retina, but it has no tendency to advance into the lens; while in choroiditis it is apt to be stellate and progressive, involving the posterior cortex after a while, and sometimes the anterior as well. When neither of these diseases can be found we have to remember that a similar opacity has been said to result from traumatism, and to be at times symptomatic of uric acid poisoning.

Anterior Polar Cataract

A small white spot is to be seen quite often at the anterior pole of the lens, perhaps so small as to be scarcely perceptible, or so large as to occupy a great part of the pupillary space. The effect on the vision varies from almost nothing to serious impairment, but a person with a large anterior polar cataract in each eye cannot see as well in bright daylight, when the pupils are contracted, as he can in the evening, when his pupils are larger.

When the opacities are flat, more or less stellate, flush with the capsule, the corneæ are normal, and the defect is present in both eyes, the condition may be pronounced congenital, and generally it is hereditary in these cases. Much more commonly the trouble is acquired, and we are able to obtain a history of a severe inflammation of the eyes. It certainly is acquired if, instead of being flat, it projects into the anterior chamber and forms a pyramidal cataract, or if a thread of connective tissue floats out from it, or joins it to the cornea, which happens in rare cases. When it is flat we examine the cornea carefully for a cicatrix, which may be hard to find even when it was made by a deep or perforating ulcer, as cicatrices sometimes clear up almost perfectly in children. As a rule the ulcer was central and perforated the cornea, but rare cases have been met with in which an anterior polar cataract resulted from an ulcer that perforated in the more peripheral part of the cornea, or did not perforate at all. The history and signs of a past iritis may suggest this as the cause. When the defect is unilateral the chances are strongly in favor of its having been acquired.

Punctate Cataract

If we look closely enough we occasionally find minute, bluish gray points scattered about in the anterior and posterior cortex, or arranged in a stellate manner about the poles. If the patient is a child these points are of little importance, as they rarely change and are supposed to be congenital, but in adults they have been observed to be the first indications of an incipient senile cataract. We do not know whether the congenital punctate spots finally prove to be centers from which a senile cataract may develop, or whether similar spots appear later in life as foci of changes in the lens.

Degenerated Cataract

Sometimes we meet with a child in whose eyes the anterior chambers are deep, the irides tremulous, the lenses cataractous and shrunken, possibly ectopic. A large part if not all of the lenticular fibers have broken down into fluid, more or less of which has been absorbed, while earthy salts may have been deposited in the lens or its capsule. The cataracts appear flat and chalky white in most cases. Sometimes they are called **milky,** sometimes **calcareous.** They resemble the Morgagnian, or hypermature senile cataracts, but are differentiated readily, not only by the age of the patient, and the history, but by their appearance. They are formed either before birth, or from congenital cataracts, while the Morgagnian is always preceded by the development of a senile cataract, and contains a hard nucleus which frequently may be seen to form a dark spot as it strikes against the anterior capsule during movements of the eye. Sometimes the fluid has disappeared so completely that the anterior and posterior capsules come nearly or quite into contact so as to form little more than a membrane; this is called **aridosiliquose** cataract if lime salts have been deposited, otherwise it often is called **membranous,** but the suggestion of *Beard* is good that it is better called **siliquose,** to distinguish it from the secondary membranous cataract which frequently follows an extraction. A siliquose cataract may be the final stage of a total or a zonular, or it may be due to either an intrauterine inflammation, or an intrauterine rupture of the capsule followed by absorption of its contents.

Beard describes as a **cystic** cataract an extremely rare form in which decomposition has occurred, so that the mass within the capsule has been rendered putrid. He says that such a cataract has something of the appearance of a deflated balloon, a part of which projects into the anterior chamber, and generally is of a yellow color. The protrusion is apt to be greater below than above, and may oscillate slightly with the movements of the head.

Traumatic Cataract

When an opacity appears in a lens soon after a known traumatism the diagnosis of traumatic cataract is easy, especially when it has developed under observation. As a rule it progresses rather rapidly

until the entire lens has become involved, but occasionally its progress is so slow that months, perhaps years, may elapse before the patient notices that his vision is impaired. The fact has been established both clinically and experimentally that a cataract may develop long after a contusion, or a dislocation of the lens, and yet be dependent on the injury. This is an important medicolegal point, and we need to know when we shall be justified in pronouncing a cataract traumatic if the history of the case is unknown or questionable.

A traumatic cataract may be caused directly by a wound of the lens, or indirectly by a contusion of the eye, a concussion of the head or body, or by an electric shock. We know that cataract can be produced by electric flashes, the X-rays, and the conditions under which glassblowers work, and it is a question whether these should be classed as traumatic or not, for we do not understand exactly how they are produced, but certainly they may be referred to external influences.

The symptoms vary with the cause, the interval of time that has elapsed since the injury, and the age of the patient. If the lens has been wounded recently we shall almost surely find a conjunctival and a ciliary injection, a hyperæmia or an inflammation of the iris, together with whatever lesions may have been produced in other tissues by the injury. The lens itself may be in its normal position, or it may be dislocated. Probably we find it swollen and occupied by a grayish white mass, part of which may protrude from a rent in its capsule. Sometimes the patient is in great pain from secondary glaucoma. If the lenticular mass is yellow, suppuration is present. In other cases the wound in the capsule is small, there is little if any protruding lens matter, and more or less of the lens may be clear. In very rare cases a comparatively small opacity may be left permanently, but, as a rule, the entire lens becomes opaque sooner or later.

In a child the lenticular fibers usually break down and are absorbed, so that at the end of a few weeks or months nothing may be left of the lens except the capsules, which have come together to form a siliquose cataract. Sometimes the wound closes before this end is attained and then we find a soft, milky looking cataract. Complete absorption cannot be expected in an adult, although it is observed occasionally even in advanced life, because the firm nucleus ordinarily refuses to disintegrate. The older the patient the more

slowly do the changes take place, but whatever his age may be a wounded lens usually becomes completely opaque. Once in a while we see a case in which a localized opacity remains stationary, extends slowly, or clears up more or less if the patient is very fortunate. Such cases are exceptional and are more common among adults than among children. Only one eye is affected as a rule in these cases, but both may suffer in some accidents, like explosions.

A cataract may develop at any age as the result of a contusion of the eye, whether the lens is dislocated or not. If a total or partial cataract forms in an eye known to have received a blow, we have good reason to pronounce it traumatic, but should this diagnosis be questioned we may need proof. If the eye is known to have had good vision prior to the accident there is no question as to the correctness of the diagnosis, but we cannot always obtain this evidence.

In a young person we need to exclude cataract that is congenital, or has been caused either by disease, or by a nontraumatic inflammation of the cornea. If the cataract is total, whitish and stellate in appearance, and the tension of the eye is normal, or perhaps slightly plus, while the other eye is normal in every respect, both the congenital form and that due to disease are excluded with a high degree of probability, because congenital total cataracts almost invariably are bilateral, or associated with defects in the other eye; while the absence of minus tension and of a past severe iritis exclude iridocyclitis as a cause, the absence of high tension and of the peculiar appearance of a glaucomatous cataract exclude glaucoma, and other diseases of the eye seldom if ever cause a total cataract. Such a very rare condition as a unilateral diabetic cataract can be excluded by an examination of the urine for sugar. If the cataract is partial and about the posterior pole we must exclude the presence of remains of the hyaloid artery and of posterior lenticonous, as well as of choroiditis, pigmentary degeneration of the retina, and the uric acid diathesis before we can positively pronounce it traumatic, but these congenital and morbid conditions are almost invariably bilateral. If the opacity is zonular in type and the other eye normal, the question is one of probability between two extremely rare conditions. An imprint of the pupil on the anterior capsule furnishes positive proof of a traumatism. A flat, unilateral anterior polar cataract probably was caused by the perforation of a corneal ulcer, just possibly by an iridocyclitis, but while it would be going too far to say

that it could not be produced by traumatism, yet the history of injury would have to be very distinct as prior to its appearance, and all other causes would have to be excluded before we could say that it was traumatic. It very rarely happens that a person is stung in the cornea by a bee, but such an accident accounts perfectly for the formation of a traumatic anterior polar cataract. Soon after the bee has stung the cornea an opacity appears in the anterior layer of the cortex of the lens in the region of the pupil, and clears up to a great extent in a few days, but usually leaves a permanent central spot. This result seems to be due to a toxic action, rather than to the traumatism itself, as it has been proved experimentally that the sting must pass through the cornea and inoculate the aqueous with its poison.

The difficulty of diagnosis is much greater when a cataract develops in an elderly person some time after a contusion, because it is hard to exclude a possible coincidental development of a senile cataract, even when it is watched from the first. This is shown by the following case. A man sixty years old was struck in the eye by a piece of wood that caused a contusion with abrasion of the skin and cornea. The pupil was dilated with a mydriatic and the lens found to be perfectly clear. Two days later converging opacities were to be seen in the upper inner periphery of the lens. An incipient cataract was evident a month later, though the vision was 20/20. The cataract progressed steadily, resembling at all times and in every respect the ordinary senile variety, while the vision fell to 20/50 at the end of three months, and to counting fingers at the end of six months. I believe this to have been a traumatic cataract because I watched its development and know the contusion to have been an adequate cause, but the possibility of a senile cataract which started just at that time independently of the injury cannot be excluded. We can only say that such a coincidence would be remarkable, and that the development of a cataract which showed its first traces in a previously perfect lens within a short time after the occurrence of an adequate injury justifies us in calling it traumatic. This case also illustrates the fact that considerable time may elapse after the receipt of a contusion before the patient notices any trouble, even when changes in the lens begin almost immediately, for the vision was 20/20 at the end of a month, and 20/50 after the lapse of three, and even a loss as great as the last often is insufficient to attract an elderly patient's attention.

A person may fall from a height and strike on his feet, or on some part of his body, may be badly shaken up, as in a railroad accident, or may receive a blow on the head, all with no contusion of the eye, and yet one or both lenses be rendered cataractous. The same thing happens sometimes after an electric shock, when the current is supposed to have passed through the eyes. There is nothing about the cataracts themselves to distinguish them from those caused by contusion, except that they are more often bilateral. They may or may not be dislocated, or associated with lesions of other tissues in the eyes. They may be complete or incomplete; in the latter case either the anterior or the posterior cortex may be affected, and they may advance to total opacification, may remain stationary, or may undergo partial involution. The same is true of cataracts caused by electric flashes, or the X-rays, which sometimes may be differentiated from those produced by electric shocks by the fact that, as a rule, they are not complicated by lesions of the deeper tissues of the eyes, while the latter are liable to be disorganized considerably by the passage of the current through them. We are not yet accurately informed how electricity produces cataract, but it would not seem to be through heat, or the coagulation of albumin, because the opacity is not formed at once, but appears after an interval.

Glassblowers are more prone than most people to cataracts that develop slowly, which may be considered in them as an **occupational disease.** The cause beyond doubt is connected with the nature of the work, but just what it is has not yet been clearly determined. Similar cataracts occur less often among ironworkers and others who have to look a great deal at bright, hot lights.

Cataract Due to Disease

We find a cataract caused by lack of nutrition in every atrophic eye. In the majority of cases it is shrunken, and either is enveloped in a mass of inflammatory exudate, or its anterior capsule is extensively adherent to the iris, and then we recognize that the nutrition of the lens and of the eye as a whole was cut off by the same disease, either a chronic uveitis, or a violent iridocyclitis. There is little danger that an iridocyclitis will induce cataract as long as the posterior synechiæ are moderate in degree, but we have grave cause for fear of such an outcome if a crater pupil, or a total posterior synechia is formed. Sometimes the inflammation subsides after the

pupil has become excluded and occluded, so that the final result, so far as the lens is concerned, may be a yellowish or grayish exudate on the capsule in the pupil, which goes by the name of **false** cataract. When the inflammation does not subside the eyeball is apt to become soft, phthisis bulbi to set in, and the entire lens gradually to become opaque. A cataract caused by a chronic uveitis begins to appear after the eyeball has become soft and begun to atrophy, and presents in the atrophic globe much the same appearance as one which has been caused by iridocyclitis.

A cataract in a moderately soft blind eye that shows no signs of a past iritis, except perhaps a few posterior synechiæ, leads us to fear that it has been caused by a total detachment of the retina.

When we see a bluish white cataract with a silky luster in an eye that has been made blind by glaucoma and is undergoing degeneration, we know it to be one of the late results of that disease and call it **glaucomatous.** An inexperienced observer may be led into an erroneous diagnosis of glaucomatous cataract in two ways:—through a greenish reflex in the pupil, which does not necessarily indicate any opacity in the lens, and through an intercurrent appearance of cataract due to some other cause. During an attack of glaucoma with greatly increased tension in an elderly person the lens is pushed forward, the pupil is widely dilated, and a greenish reflex is to be seen within the latter, which is due to the fact that the lens is sclerosed by age and so reflects light more strongly than it did when it was younger. If we look into the eye with the ophthalmoscope we find that the lens is perfectly clear, and if we dilate sufficiently the pupil of any old person with normal lenses we can obtain the same phenomenon, so this greenish reflex finally ceases to be even suggestive of cataract. The other source of error is that a traumatic or a senile cataract may develop in an eye which is suffering from glaucoma and be confounded with the glaucomatous variety, especially when we have to make the differentiation with no knowledge of the traumatism, and without having had an opportunity to watch its development. Neither the traumatic nor the senile variety presents the uniformly bluish white color, or the silky luster of a glaucomatous cataract, but a more important point is that an eye which contains the latter never has any perception of light, while in the other forms the perception may be good enough to warrant intervention, if the glaucoma is not too far advanced.

The opacities at the posterior pole of the lens met with in choroidi-

tis and in pigmentary degeneration of the retina have been discussed under posterior polar cataract, and the only thing that remains to be said is that cataracts occasionally met with in high myopia may be caused by myopic choroiditis. All of the cataracts that originate from, or are complicated by diseases of other tissues within the eye are known as **complicated,** and rarely are operable.

We can hardly say that cataract cannot be caused by almost any serious constitutional disease, for its development has been reported in connection with a multitude of such diverse diseases as typhoid fever, scarlet fever, meningitis, influenza, chlorosis, great loss of blood, rickets, and certain diseases of the skin. In many of these conditions the patients are much debilitated, and the cataracts have been called **cachectic,** but we do not know that cachexia is the cause. They are met with in persons who have suffered from convulsions, and these are classed as traumatic by some writers who believe them to be caused by the violent spasms of the ciliary muscle which are supposed to take place; but this fails to explain why such cataracts are apt to have remarkably large nuclei when they occur in comparatively young people. Diabetes alone is known definitely to induce cataract, and if we should ever see opacities develop in the lens in connection with any other general disease we should take great pains to exclude every other possible cause, and then try to discover the connecting link.

When a young person develops in both eyes within a few weeks, from no known cause, cataracts which are evenly developed, and when complete may be seen by oblique illumination to be bluish white with sectors radiating from the center that have a luster like that of mother of pearl, an examination of his urine will almost certainly reveal that he has diabetes mellitus. In these cases the lenses may become opaque in a few days or hours, but ordinarily the process is not so rapid. The patient is more likely to notice that he cannot see well and that his vision deteriorates steadily until it is reduced to counting fingers at the end of a few weeks. This condition is so characteristic in a young person that it can scarcely be mistaken, but no such characteristically **diabetic** cataract forms in an elderly patient. Cataracts appear, it is true, but they present no points by which they can be differentiated from the senile variety. We may call every cataract that develops in connection with diabetes in elderly patients diabetic, or call them all senile; no one can say positively that we are wrong, but there is one thing that can

help us occasionally. Diabetes is apt to produce a change in the clear lens such as to render the eye myopic, so we should investigate the urine of every person who becomes nearsighted suddenly from some unknown cause, and of every old person who rejoices in the possession of second sight. If such a patient is found to have diabetes, we are justified in saying that he has diabetic cataract if his lenses become opaque later.

Senile Cataract

When an elderly person tells us that he has been losing the vision of one or both of his eyes progressively for several months, although they have shown no signs of inflammation or irritation, until now it is almost gone, and we see a grayish white opacity which has a somewhat stellate structure behind the pupil and occupying its entire area, we do not need to be experts to recognize a senile cataract. It may be a diabetic cataract if the patient has diabetes, or possibly it may be traumatic, but aside from these forms the condition is so characteristic that it seems as if it were impossible to confound it with anything else. Yet mistakes do occur. The progressive loss of vision in glaucoma sometimes is ascribed to cataract, but this is only when the lens has not been examined properly and the other symptoms have been neglected. Probably the usual cause of this error is the observation of a greenish reflex from the pupil, which has already been explained as due to the sclerosis of the lens in old people and as not indicative of opacity. A pupil may be occluded by a mass of exudate that forms a false cataract, but the opacity is in the pupil, instead of behind it, and is attached to its margins, while posterior synechiæ, with perhaps other symptoms, as well as the history, testify to a violent attack of iridocyclitis. It seems almost incredible that a leucoma of the cornea should be mistaken for a cataract, yet once a patient presented a letter from a physician in which it was stated that she had a cataract and had been sent to me for an operation. She had a central leucoma of the cornea with a perfectly clear lens. There is no way to guard against the occurrence of such blunders except by training our own eyes to observe correctly.

Senile cataract almost always is bilateral, but the second eye may be affected long after the first. While there is little difficulty in making the diagnosis in the majority of cases when it is near the

stage of maturity, its recognition is by no means always easy during its early stages, when the pictures presented are different altogether from those of the final stage; so we need to investigate the lens whenever the patient has any symptoms which may be attributable to it and cannot be explained otherwise. We speak of senile cataract as being incipient, immature, mature, and hypermature, but these stages pass into one another by imperceptible degrees and are not divided by any sharp lines. An incipient cataract may be defined roughly as a lens which presents slight opacities that do not impair vision greatly, an immature as one in which these opacities have advanced sufficiently to impair the vision seriously, a mature as one in which the entire cortex has become opaque, and a hypermature as one in which degenerative changes have become manifest. An incipient cataract may progress steadily to maturity within a few months, or it may develop so slowly that years intervene between its discovery and any noticeable impairment of vision, while in still other cases we meet with periods of progression and retrogression. As a rule, there is nothing about the appearance of an incipient senile cataract from which we can determine with any confidence its probable course, so when we discover one it is not wise to mention the word cataract in the hearing of the patient, at least before it has entered the immature stage and an explanation is demanded of the progressive loss of vision, but at the same time it is well to acquaint some relative or friend of the patient with our findings and with the uncertainty of the future, if we feel that we can rely upon his discretion, in order to protect ourselves from discredit when the cataract becomes manifest.

The **subjective symptoms** of a patient with incipient senile cataract often are extremely vague. He may complain simply of a feeling of discomfort that passes away only when his eyes are closed, or he may state that his eyes tire easily while reading, and that the letters dance before him. Sometimes he is annoyed by little black specks and spots that elude him when he tries to look at them. He may have noticed that a bright light at a distance appears to be distorted, for instance that the moon seems to have horns, or that it appears to be double or multiple. Another patient seems to see through a veil; another cannot see distant objects distinctly, but has regained the power to read without glasses; in still another the vision is blurred for both distance and near. All of these symptoms may be due to changes in the lens and to their interference with

vision. We should think naturally that impairment of vision would be the first symptom to be noticed, but this is the case only when the opacity is nearly central. If a filmy cloud can be seen behind the pupil the statement of the patient that he seems to see through a veil is an accurate description. If he has recently become myopic we may discover a false lenticonus, and we examine the urine for sugar, but whether this is found or not there is grave reason to fear that opacities soon will appear in the lens. One or more opaque lines may cross the pupil in such a way as to cause monocular diplopia, or polyopia, especially when the patient looks at a bright light at some distance, or a distortion of the latter if they project into but do not cross the pupil. Small punctate opacities may cast shadows on the retina, so when a patient complains of elusive black spots we try to get him to notice whether they float about in all parts of the field, or remain in certain positions when the eye is quiet, because if they are fixed they are due to minute opacities somewhere in the refractive media, and probably in the lens. The replies to our questions often are unsatisfactory, as both forms are apt to be present and the patient's attention is likely to be attracted by the spots that make the greatest movements. The changes in the lens, even when they are very slight, sometimes produce sufficient aberration to blur the retinal images a little, and to cause weariness of the eyes through a constant endeavor to fuse a distinct with a blurred image.

None of these subjective symptoms are characteristic of cataract; all of them may be produced by other causes which are apt to suggest themselves so strongly as to lead us to neglect to make a methodical examination of the lens in many cases, particularly when we think we have discovered the origin of the trouble elsewhere. We hesitate also to dilate the pupil of an old person unnecessarily, lest an attack of glaucoma be excited, and as this dilatation is necessary to an investigation of the lens it is probably true that many cases of incipient cataract pass through our hands not only undetected, but unsuspected. It is only when the symptoms call attention directly to the lens that we are likely to investigate its condition. Before we instill a mydriatic we should always test the tension of the eye to ascertain whether the procedure will be particularly unsafe, and note the condition and reactions of the pupils. The pupils are expected to be small and to have only a moderate range of movement, but a good, prompt reaction to light will assure us of the integrity of the

iris, and probably of that of the retina and choroid. A grayish appearance of the lens may have suggested cataract, but this phenomenon is very misleading as it does not necessarily indicate an opacity. A large part of the senile lens has been changed into a hard, yellowish nucleus which reflects more light than a young lens, while the anterior capsule has acquired a dusty, grayish look; these conditions combined are apt to give rise to a grayish, or yellowish gray reflex even though the lens is perfectly transparent. After the pupil has been dilated this reflex becomes greenish because of the greater influence exerted by the yellow color of the nucleus.

We dilate the pupil with cocaine, euphthalmine, homatropine, or a mixture of the first and last, and then examine the lens by oblique illumination, afterward with the ophthalmoscope. In one case we may be able to see nothing wrong by oblique illumination, unless it may be an occasional dark line in the periphery, but when we use the ophthalmoscope by the direct method, interposing a + 10 or + 12 diopter lens, sometimes we can make out a series of clear, radiating lines that appear bright at times, and at other times cast shadows. By gently rocking the mirror, or by moving the head slightly from side to side, they may be made to seem to come and go, and we can see that they are situated just beneath the capsule in the periphery. They are supposed to be caused by water between the lenticular fibers which have not yet become degenerated, and they mark the earliest observable stage of an **incipient** subcapsular cortical senile cataract.

In a more advanced stage we may observe by oblique illumination a number of grayish, wedgeshaped opacities with their bases at the equator, their apices directed toward the anterior pole, which are developed best, as a rule, in the inner and lower part of the periphery. When they are broad and white we are led to infer that the ripe cataract will have a considerable amount of soft cortex, but when they are narrow, straight, and gray, we shall expect to find it hard. Sometimes we can infer the presence of fluid from a milky color, or from a luster like that of satin, or of mother of pearl. These details are brought out better, together with others that differ in individual cases, if we view them through a binocular loupe, or through a magnifying glass, though the latter is not as satisfactory. With the ophthalmoscope all of these opacities appear to be black against the red background of the fundus, and still more become visible. Some of them seem to move downward when the patient looks up

and are proved by this parallactic movement to be situated in the posterior cortex. The pupillary area of the lens may be perfectly clear, or it may be trenched upon by the apices of the wedgeshaped opacities, or we may be able to see fine dots and lines within it, as well as between the opacities. By rotating the mirror a little we may see bright lines come and go just beneath the capsule, as in the earliest stage, but they are located much nearer to the anterior pole. Occasionally they are to be seen in the pupillary area when otherwise that is perfectly clear, and in such cases their appearance has been compared to that produced by finely drawn out bubbles of air in glass, though they are invisible except in the way described because they differ optically from the surrounding lenticular fibers only in the index of refraction.

While using the ophthalmoscope to study the changes in the lens in a case of incipient cataract we should avail ourselves of the opportunity to investigate the condition of the vitreous, retina, papilla, and choroid, for as long as the opacities do not interfere seriously with the vision of the patient we are able to see the fundus more or less clearly, though perhaps "through a veil." In most cases we shall find a number of opacities in the vitreous, but these do not necessarily imply the presence of any grave disease.

As the opacification of the lens increases we may see the apices of the wedgelike opacities project into the pupil, or perhaps see clouds of various shapes and densities in the latter before it is dilated. The vision is not very good and we can see little if anything of the fundus, though we still obtain a red reflex. Now the cataract is **immature.** If we dilate the pupil we shall find the opacities much increased in size and number, with areas of complete opacity. During this stage the anterior chamber is apt to be rendered abnormally shallow by the swelling of the lens, but usually it regains about its normal depth as the cataract approaches maturity. The opacity of the lens grows more and more dense, the red reflex less and less distinct until it is blotted out, when vision is reduced to counting fingers at a short distance. At this time we may be able to demonstrate the cataract to be still immature by casting light obliquely on the pupil, when a shadow of the iris will be seen to fall upon the opacity if the anterior layers of the cortex are transparent.

A senile cataract is technically **mature** when the entire cortex has been replaced by a white, opaque mass in which a stellate structure usually is visible. Vision is reduced to little more than the percep-

tion of moving objects, or to counting fingers at a very short distance. An eye that cannot perceive light is blind and no good result can be obtained from an extraction of the cataract. Furthermore, the perception of light must be good in all parts of the fundus, and the patient must be able to indicate correctly the direction from which light enters the eye if we are to hope to be able to restore vision. The power to indicate the direction from which the light enters the eye is called the **projection,** and we test it by flashing a light into the pupil from various parts of the field and having the patient indicate the direction from which it comes. If the correct direction is indicated promptly, with no search for the light on the part of the eye, the projection is good and we have excluded any gross lesion of the fundus, but if the light is not seen when it is in certain parts of the field, or if it seems to come from a different quarter, the projection is faulty and an operation probably will prove a failure. Under certain circumstances sometimes it is justifiable to operate on an eye with faulty projection, but never unless the patient has been fully informed that the chance of success is small, and that the sight is likely to be destroyed sooner or later by the disease in the fundus, although the operation be successful primarily. Even when the projection is perfect the result of an operation may be a disappointment, as it is very hard to exclude the possible existence of a small patch of central choroiditis, or of a senile degeneration of the macula, both of which impair central, but not peripheral vision. Occasionally we may be led to suspect the presence of some such lesion from the strength of light needed to excite perception, but very small ones evade detection by any test.

While the picture presented by an incipient or immature senile cataract is about as described above in the majority of cases, sometimes it is quite different. The first symptom noticed may be a loss of vision, and we may be able to see by oblique illumination a smoky opacity in the center of the lens, although its periphery is perfectly clear. If this cloud occupies the nucleus we call the cataract **nuclear,** while if it is about rather than in the nucleus we say that it is **supranuclear.** In either case the opacity is seen less distinctly with the ophthalmoscope. These forms of cataract usually appear between the ages of forty and fifty and often are associated with a lenticular myopia. The opacity gradually grows denser, but the impairment of vision is out of proportion, as a rule, to the visible changes.

Occasionally when we can see little or nothing by oblique illumination the ophthalmoscope will reveal many little, round, black dots scattered through the lens, and then we may be uncertain whether we have to deal with a congenital punctate cataract, which is of no importance, or with the condition described by *Roemer* and others as the **blue punctate** cataract. The differentiation can be made only by the course, or by means of a very high magnifying power, such as is furnished in Zeiss's loupe. *Roemer* says that this instrument reveals numerous roundish, sharply defined clouds with a peculiar bluish luster in the supranuclear layers during the incipient stage, and that later numerous ellipsoidal, bluish opacities are to be seen in the anterior cortex, with their long axes about in line with the radii of the lens, not starting from the equator, and usually hidden behind the iris, but sometimes extending into the pupillary area. An immense number of minute round dots lie about and between them. Although *Roemer* pronounces this to be, next to the subcapsular cortical variety, the most common kind of cataract in old people, and says that it forms nearly half of those with which he meets, he questions the propriety of calling it senile, because its incipiency reaches back into early life, and some think it to be congenital, thus suggesting the possibility that the congenital punctate cataract perhaps develops finally into this form. It may develop at or before middle age. One of its characteristic features is the slowness with which its changes take place, another is that the anterior and posterior poles usually are the last parts of the lens to be affected. Vision fails only when the ellipsoids have become large and opacities have been added at the poles, so it may remain good for many years after the presence of a cataract has been recognized. If these observations are correct we have to hope that a more practicable mode of differentiation may be discovered, for a Zeiss's loupe is not found ordinarily in the armamentarium of a physician. They suggest the possibility that sometime we may be able to speak more confidently, through such a differentiation, than we can now concerning the time needed for the development of an individual cataract; and also that we may be guided by it in our choice of an operation, as he says that the blue punctate cataract is particularly well suited for simple extraction.

When an aged patient tells us that he can no longer see, it happens occasionally that his pupils are black, and it is not until we examine them by oblique illumination that the thought of a cataract

is suggested. We get a dark brown reflex, or sometimes one that is a little grayish, and find difficulty in seeing through the lens with the ophthalmoscope; then we know that we have to deal with a **black** cataract. The nucleus of the lens becomes yellow normally as age advances, and when the sclerosis is excessive its color deepens into brown, which impairs the vision by its absorption of the light. Sometimes we find the center of the lens to be cloudy, as in nuclear cataract, but in other cases no true opacity can be found. The entire lens has been converted into a hard, dark colored nucleus.

Still another picture is presented when a patient has had a senile cataract for a long time and it has degenerated. The color may differ little from that of a mature senile cataract, or it may be chalky white, or it may have a bluish, milky tint, perhaps with little white dots and spots scattered about beneath the anterior capsule, but we can find no trace of the stellate markings. The deep anterior chamber, probably with iridodonesis, tells us that the lens is shrunken. If we dilate the pupil a dark object may be seen in the most dependent portion of the lens, or we may see something dark strike the capsule from within when the patient makes a quick movement of the eye. Often the lens is tremulous. This evidently is a degenerated cataract, and the presence within it of a dark nucleus marks it as a **hypermature senile,** or **Morgagnian.** The lenticular fibers have been destroyed by several forms of degeneration acting together, so that the cortex has been replaced by a mass of broken down fibers, fat globules, lime, and cholesterin crystals, part of which becomes changed to fluid, which is absorbed gradually, while the lime and crystals may float about, or be deposited on the inner surface of the capsule. Exceptions are said to have been met with in which an entire senile cataract, including the nucleus, has been dissolved and has disappeared completely, leaving a siliquose or an aridosiliquose cataract, or the anterior cortex has degenerated, leaving the nucleus and the posterior cortex intact, but such occurrences are very rare.

CHAPTER XII

The Vitreous

Troubles in the vitreous are characterized by the formation of opacities which may be of nearly any size and may or may not interfere with vision. Sometimes the vision is obscured by a multitude of these opacities, each of which is so minute that it cannot be seen by itself, but taken together form a cloud. The presence of others which are too minute to be detected is inferred from the complaint of specks and hairs floating before the eyes. Other opacities are large enough to be visible with the ophthalmoscope and are either fixed, or float about. Generally speaking it may be said that any opacities in the vitreous that can be seen with the ophthalmoscope, or are in sufficient numbers to obscure the vision, are indicative of serious trouble within the eye, and that such as are visible to the patient alone may or may not be caused by any pathological change. The only congenital malformations to be observed do not really appertain to this tissue, but simply project into or through it, and are described here only because they are detected, as a rule, in the course of a systematic examination of the vitreous.

REMAINS OF THE FETAL HYALOID ARTERY

During an ophthalmoscopic examination we occasionally have the opportunity to observe a grayish tag or strand extending into the vitreous from the papilla, or from the posterior pole of the lens, sometimes joining the two. The vision may or may not be affected, but often the eyes are ill developed and amblyopic, frequently microphthalmic. Such a tag or strand is composd in most cases of the remains of the fetal hyaloid artery, or of its sheath. Rarely it contains blood and a pulsating stump projects from the papilla into the vitreous; still more rarely the blood column reaches to the lens, when we shall find on the posterior surface of the latter some remains of the fetal vascular membrane. When the vascular cord does not extend as far as the lens we should examine it carefully to

distinguish it from another congenital anomaly in which a loop starts from the central artery in the papilla, extends out into the vitreous and then returns, the ascending and descending portions twisting about each other in their course. This does not seem to be related in any way to the hyaloid artery, though it may readily be mistaken for it, but is simply an anomaly in the course of a branch of the central artery. It has no effect upon the vision or on the well being of the eye.

The only other congenital anomaly that we are at all likely to see in the vitreous is the presence, in microphthalmic eyes, of bands or masses of fibrous tissue, which usually are associated with remains of the hyaloid artery.

MUSCÆ VOLITANTES

Often a patient is distressed because of one or more spots of fantastic shape that move about in his field of vision. He may have been reading or writing when his attention was attracted by a peculiar spot that floated down near his line of vision, moved out of sight when he tried to look at it, and reappeared as soon as he returned to work. Probably he noticed several of these spots. We find the eyes perfectly healthy, his vision good, can detect no opacities anywhere in the refractive media, and are able to assure him confidently that the spots are in no way symptomatic of any disease which threatens the integrity of the eyes. We call them muscæ volitantes. They are supposed to be **shadows** cast by microscopic particles in the vitreous, perhaps wandering leucocytes, massed débris of cells, or possibly minute detached filaments of tissue. They may appear suddenly and persist for many years without apparent change, or they may appear and disappear from time to time, and many persons are able to detect them at any time that they wish to look for them. Their appearance seems to be more noticeable when the eyes are strained or overworked, and in conditions of bodily weakness or fatigue, so it is possible that such conditions render the perceptive apparatus hypersensitive to the shadows cast by these minute defects in the vitreous. This idea receives some support from the facts that the persons who observe them most commonly are myopic, or are accustomed to use their eyes a great deal in fine or near work, and that often they can be made to disappear by rest, the administration of tonics, and the correction of refractive errors, provided that the patient ceases to pay any attention to them.

Muscæ volitantes must be differentiated from true opacities, whether fixed or floating. When a patient tells us that while he is looking quietly at a certain point in a white surface a black spot is always present in exactly the same relation to his visual line, the spot must be caused by a fixed opacity somewhere in the refractive media, and probably we shall find it in the lens, so the first thing we have to do is to have the patient observe whether any of the spots of which he complains keep a fixed position when his eyes are quiet. If none of them do this we can dilate the pupil so as to get a clear red reflex from the fundus with the ophthalmoscope held several inches away from the eye, have the patient move his eyes quickly up and down a few times and then hold them quiet. If no black specks can be seen to sink across the red reflex we know that no visible floating opacities are present in the vitreous, and, as the eyes are healthy and the vision good, the diagnosis of muscæ volitantes is positive.

FLUID VITREOUS

If on the contrary we see black opacities sink across the red reflex we know that we have to deal with a pathological condition, and that the vitreous is more or less fluid. Almost invariably the vision is impaired to some degree in these cases. The degree of fluidity may be estimated roughly from the rapidity with which the opacities move; if they pass rapidly across the field the larger part of the vitreous is fluid, but if they move slowly the liquefaction is slight, or not so far advanced.

When the nutrition of the vitreous is impaired by inflammatory or other causes it tends to become fluid, with the formation of opacities that are shaken about in the fluid by the movements of the eye and sink to its lowest part when the organ is at rest. The presence of floating opacities leads us to investigate closely for their cause, as they are met with during and after **inflammation of the uvea,** especially choroiditis, in some cases of retinitis, in high myopia, after hemorrhages, and after wounds of the eye. In these cases we need to take careful note of the tension of the eye from time to time, and to observe whether there is any increase in the rapidity of the movement of the opacities across the field. The tension of the eye may be normal, or it is increased when glaucoma is present, but generally a soft eye is an indication that the vitreous is fluid. An increasing fluidity, with a tension which is continually becoming lower, after any

inflammation is a bad prognostic sign, as it indicates the onset of atrophy, but atrophy is not to be feared as long as the tension remains normal.

When we find little opacities that float slowly in the eye of an elderly patient it is well to examine his lens in search of an **incipient cataract,** as such floating opacities frequently accompany that condition. The reason for their appearance is not understood, but they do not seem to be in any way a bad prognostic sign.

In other old people, sometimes after the extraction of a cataract, we see opacities that reflect the light and look like brilliant spangles as they dance about or sink through the vitreous. This is called **sparkling synchysis,** or **synchysis scintillans.** The opacities are formed of crystals, mainly of cholesterin, but whence they originate, or how they come to be formed in the vitreous, is unknown. Usually the eyes otherwise are healthy.

HYALITIS

When the vitreous is ***very hazy*** we are accustomed to say that the patient has hyalitis, although there is no such thing as an inflammation of the vitreous. The cloudiness may be so dense that we can see only a red reflex from the fundus, perhaps not even this, but in most cases we are able to see the details of the fundus indistinctly, while the papilla looks unusually red. Not infrequently the picture presented by the papilla resembles closely that of a neuroretinitis, but we are able in most cases to make the differentiation at once by turning our attention to the periphery of the fundus, for this appears normally clear, as a rule, in neuroretinitis, while it is obscured equally with the center in hyalitis. The cloudiness is due to the presence of an immense number of opacities which are so minute that no one of them can be perceived with the ophthalmoscope, although together they obstruct the vision. When they are comparatively few they are apt to be very difficult to detect, for then they may scarcely interfere with the transparency of the vitreous.

In other cases of hyalitis we see filmy patches in the diffuse haziness, or distinct opacities here and there. Occasionally the latter may be very large, and some of them may adhere together so as to form membranes. In rare cases a dull yellow reflex from within the eye informs us that suppuration is present, and that the patient has an abscess of the vitreous, or a **suppurative** hyalitis. This is to be dif-

ferentiated from a glioma of the retina and a pseudoglioma, both of which give a yellow reflex from deep in the eye, by the history of a wound, or the presence of some septic disease or focus from which an abscess has started by metastasis, together with the age of the patient, and the history.

With the recession of the primary inflammation an ordinary hyalitis may clear up, the opacities become absorbed, and the vitreous regain its transparency, or the latter may become more or less fluid with permanent floating opacities.

HEMORRHAGE INTO THE VITREOUS

When an eye has suffered a great loss of vision, either spontaneously or after a traumatism, and we can get little or no red reflex from the fundus, but by oblique illumination can see a red mass, or at least a reddish appearance behind the lens, we know that a very large hemorrhage has taken place and occupies a great part of the vitreous. In another patient we may see a black mass with red borders, or a red mass lying in front of the retina. In still another, whose vision is not quite so badly impaired, we may see a bright red film between us and the fundus, and, when this film lies in front of the macula, the patient may state that objects seem to him to have a reddish color. Occasionally, but not often, we can see the place where a retinal vessel has been ruptured.

Our most important duty in such a case is to learn the cause of the hemorrhage. This is self evident when the eye has been wounded, or has received a blow, but when the hemorrhage has occurred spontaneously, or has followed a severe bodily exertion, the cause must lie in an abnormal condition of the blood vessels, or of the blood itself, and can be traced to a general or a local disease. Arteriosclerosis is the cause in many old people, but we should always make a thorough examination to exclude more active diseases. The walls of the vessels may have been weakened by syphilis, or by an excessive indulgence in alcoholic liquors. Both the urine and the blood should be examined, for the patient may be found to be suffering from diabetes, or perhaps from renal disease, and such hemorrhages occur in anæmia, chlorosis, and leucocythæmia. They have been observed also in pregnant women. Sometimes they occur in emphysema of the lungs, when it seems to be probable that the respiratory effort serves to rupture a blood vessel which has been weakened by some

other disease. When no other cause is apparent we should remember that the claim has been made that a hemorrhage into the vitreous may be the first sign of a tuberculosis of the ciliary body.

Such local diseases as high myopia, choroiditis, and retinitis furnish an adequate explanation of a hemorrhage into the vitreous when they are known to have been in existence in the eye prior to its occurrence, and when other evidence of the presence of such a disease is clearly manifest, but when the previous history is unknown and the hemorrhage is large, we frequently are obliged to wait until at least a part of the blood has been absorbed to establish such a cause.

These hemorrhages are absorbed very slowly. The vitreous itself becomes more or less fluid, and a part of the blood may remain for a long time in the form of large or small floating opacities. Sometimes the hemorrhages recur several times, and then connective tissue is apt to develop, and produce the picture of proliferating retinitis, perhaps with a new formation of vessels.

Recurrent Hemorrhages into the Vitreous

Occasionally we meet with a young man who presents a peculiar clinical picture, the cause of which is unknown. At irregular intervals hemorrhages take place into the vitreous, often coincidentally with nosebleed. Sometimes he gives a history of having been engaged in violent exercise at the time the hemorrhage occurred, but the bleeding seems to be quite as likely to happen when no such explanation can be furnished. The young men may be in almost any kind of physical condition from hearty and robust, to weak and anæmic, and may or may not be suffering from disease. A striking feature in these cases is that the blood is absorbed rapidly, instead of slowly as in other forms of hemorrhage into the vitreous. The worst feature is that after a number of recurrences a proliferating retinitis, in the form of a growth of connective tissue in the vitreous, usually accompanied by many new vessels, is likely to develop and to impair the eye permanently.

Preretinal Hemorrhages

A red disk that is either round or has a horizontal upper border, and lies in front of a part of the retina, is a hemorrhage situated between the retina and the vitreous. In most cases it covers the

macula, rarely the papilla. It is met with most often in young persons, and, as a rule, the blood comes from the retinal vessels.

FOREIGN BODIES AND PARASITES IN THE VITREOUS

When we see a part of the vitreous encroached upon from any direction we have to differentiate between a tumor of the retina or choroid, and a detachment of the retina. When an eye has been wounded a foreign body may be seen sometimes hanging in the vitreous, perhaps surrounded by a cloudy areola. Very rarely, at least in this country, we may see something that looks like a bluish white bladder in the vitreous, perhaps attached to the retina, which is recognized to be a cyst. If movements of its neck can be seen it may be diagnosed as a **cysticercus,** which is the probable diagnosis whenever a cyst is seen. Its appearance has been preceded by a detachment of the retina, the cause of which could not be recognized until the cyst broke through, and sooner or later the vitreous will become turbid and the eye be lost through an iridocyclitis. In certain parts of the world filariæ are said to form colonies in the vitreous that impair the vision but otherwise cause little trouble until they attack the ciliary body, or enter the anterior chamber, where they excite much pain and sometimes inflammation.

CHAPTER XIII

Abnormal Tension of the Eyeball

Although the tension of the eye varies within rather wide limits we need to be able to recognize the condition when it is either above or below normal, for both an increase and a decrease are pathological symptoms. The ***simplest and most useful method of testing the tension*** is to have the patient look down, to place the tips of the two forefingers on the upper lid near the margin of the orbit, and to press with them alternately upon the eyeball, just as we examine a swelling for fluctuation. The sense of touch has to be educated by testing the tension of many normal eyes in this manner, and the physician soon learns to detect any marked variations, although slight ones may evade him because he can have no absolute standard as a basis for comparison. We should compare the tension of the two eyes in every case, and we press more firmly than usual when the lid is œdematous, as the œdema imparts a cushiony feeling of softness to the finger. Following the suggestion of *Bowman* we designate normal tension as Tn, a questionable rise as + ?, a slight but distinct rise as + 1, a considerable increase as + 2, and stony hardness as + 3. Decreased tension is designated in like manner as — 1, — 2, and — 3. According to *Schioetz* the tension varies normally between sixteen and twenty-seven millimeters of mercury, and can be measured more accurately by the **tonometer** devised by him, or perhaps still better by the modification proposed by *Gradle*. This instrument does not supplant, but it supplements the use of the fingers, as its systematic employment enables us to judge somewhat concerning the state of affairs within the eye, especially when the tension is increased. Its measurements are not absolutely accurate, so it should be applied three times in each examination and the readings averaged. After the eye has been anæsthetized with a two per cent. solution of holocaine the patient is placed on his back, his head bent rather backward so that his eyes are directed upward, he is told to look straight up, the lids are

separated with care to make no pressure on the eyeball, the instrument is placed on the cornea, and the number of millimeters of mercury necessary to indent it is read on the scale. Weights have to be used in addition if the tension is plus, and then the tension is determined in millimeters of mercury by reference to a chart.

GLAUCOMA

Of all the diseases of the eye glaucoma probably is the one most often mistaken for some other, more curable condition, and such an error is almost invariably attended by disastrous consequences. Many years of experience have shown that when this disease has once become firmly established we may delay its course and enable its victims to retain some sight for a good while, as long as they live in many cases, for the majority of the patients are elderly, but that it continues to progress either slowly or rapidly in spite of all that we can do. An early diagnosis is extremely important because a cure can be effected only when treatment is instituted during an early stage, and yet the fact that it is made so seldom, especially by those who see comparatively few cases of the disease, is not a matter of surprise when we consider the wide variations in the subjective and objective symptoms which are presented in its different forms, and the resemblance these symptoms bear to those produced by other conditions of the eye, as well as occasionally to those which are characteristic of certain general diseases.

The **symptoms** common to all forms of glaucoma are a loss of sight, which may be rapid or very slow, an increase of tension that ranges from the upper limit of normal to stony hardness of the eyeball, an excavation of the papilla that is diagnostic when fully developed, and a contraction of the field of vision which begins and is most pronounced on the nasal side. The combination of any two of these symptoms may suffice for the diagnosis, but the symptom which is the most characteristic at all stages and in all forms of the disease is an increase of tension. Even when the increase is not apparent to the palpating fingers, it may be demonstrated to be present, at least at intervals, by systematically repeated examinations with the tonometer. The tension should be tested with the fingers as a routine part of the examination of every eye, and when this is supplemented by a test of the vision, a perimetric examination of the field, and an ophthalmoscopic examination of the in-

terior of the eye, there is little danger that a case will escape our observation.

A glaucomatous **excavation** begins sharply at the edge of the papilla, which sometimes is surrounded by a yellowish gray ring, the so-called halo, and we must be careful not to mistake this for a part of the papilla itself. In most cases we can make out a line of separation between the papilla and the halo because the two differ slightly in color, and sometimes the line is very clear because of the presence of a scleral ring. The vessels hook over the margin of the papilla and do not seem to be continuous with those that lie in the floor of the excavation. As long as the excavation is shallow the color of the papilla may be good, but as it becomes deeper the color changes to bluish or gray, and when it is very deep dots that mark the apertures of the lamina cribrosa may be seen in the floor. The retinal veins are broad and tortuous, the arteries are apt to be engorged and to pulsate at first, but later they become small. These are the essential points that mark an excavation of the papilla to be glaucomatous, but the different forms of excavation that are met with and their differentiation will be given in the study of the fundus.

Glaucoma is **secondary** when it occurs as a complication of some other lesion of the eye, and is unilateral in most cases. Hemorrhagic glaucoma may be considered a variety of the secondary. Primary glaucoma comes on without known cause as acute inflammatory, chronic inflammatory, simple, and infantile, the symptoms of which differ so widely that at first we are apt to find it hard to believe them varieties of the same disease, but all terminate in absolute glaucoma, and are very closely related.

Prodromal Symptoms of Glaucoma

It is unfortunately the fact that while the premonitory or prodromal symptoms which usually precede an attack of acute glaucoma may attract the attention of the patient sufficiently to cause him to consult a physician, they are often considered by him to be of no importance, and consequently the disease is neglected at the very time it is most susceptible to treatment. When an elderly patient suffers from facial neuralgia or headache, especially if it is associated with loss of sleep and of appetite, it is worth while to examine the eyes. The neuralgia usually affects the first or oph-

thalmic branch of the fifth nerve, but the second branch also may be affected and the trouble may seem to be situated in the teeth. The toothache has been so marked in some cases that healthy teeth have been drawn in the hope of obtaining relief when the trouble really was in the eye. In other cases this pain is not severe and an observant patient may have noticed that objects have seemed misty at times, or that rainbow colored rings have appeared about lights, but such information is not volunteered very often, and we must avoid direct or leading questions in regard to such visual symptoms because many patients are led to believe that they have had them after they have been suggested. These prodromal symptoms are apt to make their first appearance during a period of mental stress or of bodily fatigue, and to pass away in a few hours, which is one of the reasons why little attention is likely to be paid to them. In still other cases the only premonitory symptom of the onset of glaucoma seems to be an unusually frequent change of presbyopic glasses.

None of these symptoms are necessarily indicative of a threatened attack of glaucoma, but when they happen to be, and we examine the eye while they are present, we find the tension increased, usually about + 1, and can see by oblique illumination a slight ciliary injection, a slight diffuse cloudiness of the cornea, a rather shallow anterior chamber, and a dilated pupil which does not react well to light. With the ophthalmoscope we may see a venous engorgement and an arterial pulsation of the retinal vessels, but no typical excavation of the papilla. If the attack happens to be the first we are not likely to be able to find anything abnormal in the eye after the symptoms have passed off, but, if we can find no evident cause for the symptoms elsewhere, our suspicions should be aroused, and the patient should be urged to have an immediate examination in case he ever has a second attack. We cannot tell at this time whether the disease will assume the acute, or the chronic inflammatory type, but an iridectomy probably will effect a permanent cure.

Acute Inflammatory Glaucoma

Should the disease assume the acute form the patient will be seized at some subsequent date with intense pain radiating through one side of the head, nausea, vomiting, and prostration, usually during a period of weakness following unusual fatigue, great mental

anxiety or shock, or some other debilitating influence, like hemorrhage. These symptoms are apt to be so pronounced as to suggest trigeminal neuralgia, migraine, meningitis, gastric trouble, influenza, or some other disease. The patient may or may not notice that the pain starts from the eye, and if he does not this organ is quite likely to escape attention unless we examine it habitually in such cases. If we find the upper lid œdematous, the eyeball reddened, the conjunctiva perhaps chemotic, and sometimes with a little discharge, we know that the eye is either the seat of the trouble, or is suffering from an intercurrent affection. The only ocular conditions that can give rise to such grave general symptoms are a violent orbital cellulitis, a severe iridocyclitis, and acute inflammatory glaucoma. If there is exophthalmos we look for other symptoms of the first, if there is not, a cellulitis is excluded. If the tension of the eye is about normal, the pupil small and irregular, the iris muddy with many posterior synechiæ, and little deposits can be seen on the lower part of Descemet's membrane, the disease is iridocyclitis, but if the tension is + 2 or + 3, the pupil dilated and oval, the cornea anæsthetic and hazy, and the anterior chamber shallow, we have to deal with an acute inflammatory glaucoma. If there is no exophthalmos, the tension is normal, and the symptoms of an iridocyclitis are absent, the affection of the eye is intercurrent, and is not the cause of the general symptoms.

When the tension is + 3 we cannot dent the eyeball with the finger, the cornea is anæsthetic and very cloudy, the anterior chamber is almost obliterated, the oval pupil is dilated extremely, the pain is atrocious, the vision is very poor and in imminent danger of being lost. In the rare cases of **fulminating** glaucoma the vision may be destroyed within a few hours, the attack may not have been preceded by any premonitory symptoms, the pain is excruciating, and the violence of all of the acute inflammatory symptoms is extreme. The symptoms abate after a while in the majority of cases of acute inflammatory glaucoma, and the vision improves, but it never again becomes as good as it was before the attack. Certain signs are left behind from which we can infer what has taken place and recognize the predisposition of the eye to further attacks. The anterior ciliary veins remain distended, the anterior chamber remains shallower than it should be, while the pupil has at least a tendency to dilate, and reacts more slowly to light than is usual in persons of the same age. The papilla is apt to appear much reddened after

the first acute attack has passed off, but not to present a typical excavation, and an operation at this time is likely to be permanently successful.

Unless interrupted by operation the course of an acute inflammatory glaucoma is that the symptoms may or may not pass away completely. In the first case recurrent attacks, which, as a rule, are not as severe as the first one, take place at intervals, but the vision is left worse after each, a glaucomatous excavation forms in the papilla, and finally the eye is rendered totally blind. In other cases the first attack cannot be said to pass off entirely; the symptoms are ameliorated, but persist, the tension remains above normal, and chronic inflammatory glaucoma supervenes.

Chronic Inflammatory Glaucoma

Although this form may set in with an acute attack, as has just been stated, it cannot be said to do so as a rule. Many aged patients are not aware that they have any disease of the eyes, although they have had recurrent attacks of supraorbital or facial neuralgia, and have noticed at times that they could not see well. They have ascribed these symptoms to various causes, frequently to the fact that they were growing old, which also sufficed to explain to them why they have had to change their reading glasses so often. Yet these are the ordinary subjective symptoms of chronic inflammatory glaucoma.

This form of the disease begins like the prodromal stage of the acute with an attack of supraorbital or facial neuralgia, and a cloudiness of the cornea which causes hazy vision and colored rings about lights to be noticed by observant patients. This passes away in a few hours, and another attack ensues some weeks or months later, which likewise proves transient and generally leaves the eye in good condition. This is succeeded by others, with the intervals between them growing shorter constantly, and after a few have occurred permanent abnormal changes will be found to have taken place in the eye. Between the attacks we find the vision more or less impaired, the tension above normal, dilated, tortuous veins at the insertions of the muscles into the sclera, a shallow anterior chamber, a dilated pupil which does not react well to light, a contraction of the nasal portion of the field, and a glaucomatous excavation of the papilla, and we find all of these symptoms to be intensified after

each successive attack. It frequently happens that as long as only one eye is affected the patient does not realize that there is anything the matter with his eyes, and it is only after the second has been attacked and its vision fails that he seeks our aid in his search for a pair of glasses to enable him to read with comfort. Often much valuable time is lost through visits to opticians and optometrists, who are extremely unlikely to be able to recognize the presence of the disease, for if such cases can be operated on early enough a cure may be effected, or the progress of the disease delayed sufficiently to give the patient vision for the rest of his life, while if too great an advance has been made the best we can do is to delay its progress as long as possible.

When the disease is far advanced the vision is very poor, the eye has a peculiar dead appearance, broad, dark vessels ramify over the sclera and form a plexus about the cornea, the cornea is somewhat anæsthetic and not perfectly clear, the anterior chamber is very shallow, the pupil is widely dilated, oval vertically, and reacts to light slowly, or not at all, the iris is atrophic, and we get a greenish reflex from the lens. This greenish reflex is not a sign of cataract, as it has often been taken to be, but is to be obtained from all clear senile lenses when the pupils are dilated. The tension is distinctly elevated, the field is contracted, particularly on the nasal side, the papilla is surrounded by a halo, is bluish or gray and deeply excavated, the vessels hook over its margin, and their entrance into the nerve may be seen to be pressed over to the nasal side. The prognosis in this stage is bad.

An eye which is suffering from chronic inflammatory glaucoma is liable to an acute attack, and as acute glaucoma sometimes passes over into the chronic form it is evident that these two diseases are closely related, and differ in the intensity of their symptoms, rather than in their nature. The essential features in both, from first to last, are an abnormally high tension, progressive loss of vision, contraction of the nasal field, distention of the ciliary veins, abnormal shallowness of the anterior chamber, dilatation of the pupil, and excavation of the papilla.

Simple Glaucoma

While the inflammatory varieties of glaucoma can be recognized with comparative ease, if we always investigate the condition of the eye in every case of mild or severe trigeminal neuralgia with which

we meet in elderly patients, our attention seldom is called to a case of simple glaucoma in its early stage, as the only **subjective** symptoms are likely to be a gradual loss of vision and a frequent change of presbyopic glasses. Externally the eye usually appears to be normal. The cornea is clear and sensitive, the anterior chamber is normally deep, the pupil is of its usual size and reacts promptly to light, and the tension does not seem to be increased, but we must never hazard an opinion when the vision is below normal without an ophthalmoscopic examination. Above all we must never be misled by a gray reflex from the pupil into thinking the case to be one of incipient or immature cataract, for such a reflex often is seen in the eyes of old people who have perfectly clear lenses, and is caused by the reflection of light from the hard nucleus, together with a dusty, grayish look that the capsule is apt to acquire with age. Many a patient has slowly and irretrievably lost his sight while waiting for a supposed cataract to mature.

When the vision remains subnormal after the refractive errors have been corrected with glasses, the diagnosis of simple glaucoma is made readily if there is a glaucomatous excavation of the papilla, but in an early stage of the disease this may not be typical. If no other disease can be detected to explain the loss of vision, and there is no typical excavation of the disk, we have to study the retinal vessels, the field of vision, and the tension of the eye, before we can exclude or diagnose this disease. Ophthalmoscopically we notice whether the retinal veins are larger than normal, whether the arteries can be made to pulsate by a slight pressure on the eyeball, whether a vessel forms a hook as it passes over the margin of the papilla, if any of them seem to be broken at the margin, if those in the papilla lie at a lower level than those in the retina, and if the entrance of the vessels seems to be pushed over to the nasal side. With the perimeter we ascertain whether there is a contraction of the nasal portion of the field. With the tonometer we test the tension every two or three hours to learn whether it fluctuates and ever rises above the normal. If any such conditions of the retinal vessels, or of the papilla, a contraction of the nasal portion of the field, and occasional rises of the tension above normal are detected, with nothing apparently to account for any of them, the diagnosis of simple glaucoma is to be made.

In the early stage of simple glaucoma we find a loss in the nasal portion of the field alone, later there may be concentric contraction,

or sectional defects, but generally the contraction on the nasal side remains prominent. The color fields are affected in about the same way as that for white.

A study of the tension with the tonometer in this disease tells us that though it may not seem to the fingers to be elevated, it is not necessarily normal. We do not know the exact limits within which the tension can fluctuate and still be physiological in any individual eye. What we accept as the upper limit may be much too high in an individual case, and then an important symptom is overlooked. When we measure the tension every two or three hours with the tonometer we shall find in some cases no fluctuation that we can call abnormal, while in others it runs up to or above the limit, sometimes to + 1, in the morning and evening. Similar rises may be induced by excitement and other causes. At such times we may be able to demonstrate a transient obscuration of vision, or some of the other prodromal symptoms already described, but they are not very marked.

The great majority of cases of simple glaucoma are bilateral, though the disease is confined to one eye more often than in either of the inflammatory forms. Rather frequently we meet with a case that seems to be on the border line between simple and chronic inflammatory glaucoma. The eye at times presents an increase of tension with pain, or at least discomfort, distended ciliary veins, a steamy cornea, a shallow anterior chamber, and a dilated pupil, while at other times it exhibits none of these symptoms. Such a case occasionally becomes distinctly one of the inflammatory variety, and the close relation of simple to these forms is further shown by the fact that acute glaucoma may set in at any time.

Infantile Glaucoma

When the eye of a child is attacked by primary or secondary glaucoma the picture produced is quite different from that ordinarily seen in an adult, and is so characteristic, when fully developed, that there is little danger of an error in diagnosis. The eyeball protrudes because it is enlarged in all of its diameters, its sclera is bluish, its corneoscleral margin is ill defined, its cornea is very large and abnormally curved, perhaps is clear, perhaps is more or less opaque. This condition is called **hydrophthalmos** and **buphthalmos,** terms which are synonymous with infantile glaucoma. Very

few cases indeed are met with in which the resistance of a child's sclera is sufficient to enable the picture of adult glaucoma to be formed.

In the early stage of this disease, before the globe has become enlarged, the cloudiness of the cornea may suggest interstitial keratitis, but the differentiation is made instantly by a test of the tension, which is elevated in infantile glaucoma, but is about normal in keratitis. Later the protrusion of the eyeball may be mistaken for exophthalmos, so we need to notice whether the eyeball is of normal size and is pushed forward in its entirety, or is enlarged in all of its diameters, as well as to test the tension.

When the eyeball is much enlarged and the media are clear we find the anterior chamber very deep, the iris large, dull, atrophic, and tremulous, the lens apparently much too small, and the papilla with a typical glaucomatous excavation. The lens, which really is of about normal size, may be held in place by a zonule that evidently is greatly stretched, or it may be luxated backward or forward because the zonule has ruptured. These changes are all due to the fact that the tissues of a child's eye are more elastic than those of an adult, and yield before the increased intraocular pressure. The sclera and cornea give way in all directions, the other coats of the eyeball, together with the iris and zonule, are stretched at the same time, and the papilla becomes excavated, while the lens alone remains unaffected, or is compressed.

The primary form of infantile glaucoma almost invariably is bilateral, the secondary may affect either one or both eyes. The commonest causes of the secondary form are corneal ulcers and traumatism, those of the primary are uncertain. Heredity seems to play a part in the production of the latter, which is apt to accompany neurofibromatosis of the lids and orbit and so seems to be connected with a disease of the ciliary nerves, but the sinus of the anterior chamber has been found to be closed by an adhesion of the root of the iris to the cornea, and not infrequently we find that Schlemm's canal is absent.

Secondary Glaucoma

When a patient who is suffering from any disease or injury of the eye complains of pain, or an increase of pain, we test the tension at once, and if it is increased we say that he has secondary glaucoma.

Sometimes other confirmatory symptoms can be obtained, but in many cases the rise in tension is our sole reliance, because of the conditions created by the primary trouble.

The commonest causes of secondary glaucoma are ectasiæ of the cornea or sclera, adherent leucomata, extensive posterior synechiæ, swelling or dislocation of the lens, intraocular tumors, and intraocular hemorrhages, but this affection is met with in a great variety of other diseases, including contusions, and *Roemer* goes so far as to say that it may occur in almost every serious disease of the cornea, iris, lens, retina, and choroid; so we should keep a close watch over the tension in every case of ocular trouble with which we have to deal. Sometimes it follows an operation on the eye, like a cataract extraction, when it is apt to be due to an inclusion of the iris in the cicatrix, but it may be the result of other causes, one of which is a proliferation of the corneal epithelium through the wound so as to form a cyst that occupies the anterior chamber and blocks the outlet of the aqueous.

A ***dilated pupil*** in an elderly patient always is suggestive of glaucoma, but is not diagnostic by itself. We test the tension and look for some other cause when this is normal. Bilateral immobile pupils suggest a paralysis caused by diphtheria, lead or ptomaine poisoning, or syphilis, one eye alone affected in this way suggests a contusion, a lesion of the oculomotor nerve, or the instillation of a mydriatic, and we make the differentiation from the history with the concomitant symptoms. If there is no indication of any of the diseases mentioned, the tension is about normal, and the pupil is absolutely immobile, the dilatation must be due to a contusion, or to the instillation of a mydriatic. If it is recent and there are no signs of a blow, we are constrained to believe that a drug has been instilled. Some patients are ashamed to admit that they have experimented with such a drug, but we should not refuse to believe their denials until we have proved the contrary by exclusion, and one thing we have to exclude is glaucoma. Sometimes we find the tension elevated, but not enough to account for the dilatation, when the pupil is absolutely immobile, because absolute immobility is met with only in the late stage of glaucoma, or when the tension is + 3.

We need to use cycloplegics in certain diseases of the eye, sometimes for diagnostic purposes, whatever may be the age of the patient, but we should remember that they *tend to increase the tension,* and that this increase may exceed the normal limit in old people so

as to excite a secondary acute or subacute glaucoma. It is questionable whether we should call every increase of tension incident upon the instillation of a cycloplegic glaucomatous, especially as the majority of them prove to be transient, are not accompanied by other symptoms, and do no harm. A similar increase of tension accompanies every irritant lesion of the trigeminus, cyclitis, and, more transiently, irritation of the sympathetic. The difficulty is to know where to draw the line, and perhaps it may be safer to consider every increase of tension to be a symptom of glaucoma, because the other symptoms follow when it has reached a certain point.

Hemorrhagic Glaucoma

An attack of glaucoma that supervenes upon an intraocular hemorrhage is called hemorrhagic. Intraocular hemorrhages occur occasionally during or after attacks of glaucoma, but do not change the nature of the disease to this type. The differentiation between these two conditions is important to the treatment and prognosis, as well as sometimes for medicolegal reasons.

The diagnosis is not difficult when the patient has been under observation and is known to have suffered from sclerosis of the retinal vessels with hemorrhages, or perhaps from an albuminuric retinitis, prior to the onset of an acute or chronic inflammatory glaucoma, or to have had glaucoma before the hemorrhages occurred, but it is not so easy when the history is unknown.

We find hemorrhages in the retina or vitreous, some perhaps dark and old, some red and fresh. The papilla may be red and hazy, sometimes swollen, at other times excavated, and the excavation may or may not be filled with lymph and blood. We study the lesions to determine if possible their relative age. If the papilla is red, hazy, and not deeply excavated, while evidences of old hemorrhages, such as dark masses, white spots, or proliferations of connective tissue can be found, we pronounce the case unhesitatingly to be one of hemorrhagic glaucoma; while if the glaucomatous symptoms indicate an advanced stage of the disease, and the hemorrhages are fresh, we feel equally confident that the latter have appeared as a complication. In other cases the relative ages of the lesions are uncertain and we remain in doubt. Sometimes after an acute glaucoma has subsided sufficiently for us to examine the fundus, we see little linear or blotlike hemorrhages that radiate out from the papilla, and we

cannot tell whether they are the cause or the result of the glaucoma, though if other symptoms of retinitis or neuroretinitis are present we are justified in assuming them to be the cause.

Absolute Glaucoma

All of these forms terminate in absolute glaucoma. When a blind, painful eye, which is as hard as a stone, presents a bluish white sclera traversed by many large, bluish red vessels that form a wreath, the **caput Medusæ,** about the cornea; a cornea which is more or less clear, is anæsthetic, and has lost its sharp differentiation from the sclera; an abolished anterior chamber; a pupil that is dilated so widely that the irides are reduced to narrow, atrophic strips pressed against the posterior surface of the cornea, the diagnosis is easy. The eyeball passes next into the condition of **glaucomatous degeneration,** during which the patient is apt to have luminous sensations that delude him into a hope that he may yet have his sight restored. The changes in the cornea are the most noticeable; ribbon-like opacities may form horizontally across it, the epithelium may be detached as bullæ, or the tissue may break down in ulceration, and the lens becomes cataractous. From this point the course varies. Some globes undergo phthisis, with or without perforation of an ulcer of the cornea, others become enlarged in all their diameters as a result of the degenerative softening of the capsule, in others staphylomata are formed between the cornea and the equator.

SUBNORMAL TENSION

When we find the tension of an eye to be subnormal we know that there has been a decrease in its volume, caused either by a loss of a portion of its contents, or by an interference with the secretion of the intraocular fluids. It is true that the tension may be reduced somewhat by a tight bandage, through the increased outflow of fluid probably, or by the action of certain drugs, especially when a solution of one of them is instilled into the conjunctival sac, but the reduction thus produced is temporary and is supposed never to fall below the normal limit of the individual eye. Ordinarily we find a local cause for a very marked reduction of the tension, but occasionally it is a grave disease of the general organism, like diabetic coma, or a lesion of the sympathetic nerve, and the eyeball becomes

quite soft after death, as well as after enucleation. An abnormally low tension is borne by the eye without definite injury for a much longer time than one which is correspondingly high, but in many cases it is a symptom of atrophy of the globe.

After an operation in which the eyeball is opened, like cataract extraction, iridectomy, or trephining, the tension is expected to become abnormally low at once, and to regain its level with the closure of the wound and the reformation of the aqueous; the persistence of low tension with a nearly empty anterior chamber leads us to suspect the presence of a fistula, or of a cystoid cicatrix, through which the aqueous drains away too rapidly. An alternation of this condition with one in which the tension is somewhat higher, and the anterior chamber partially filled, points to abortive attempts on the part of a fistula to heal in these cases, as well as in those in which an ulcer has perforated the cornea.

Frequently we find the tension reduced somewhat after a contusion, or a slight injury to the cornea, as well as in various forms of keratitis, but, as a rule, in these cases the symptom is not of serious import. A low tension is present regularly in iridocyclitis, and in other forms of uveitis in which the outlet of the intraocular fluids is not blocked, and then it furnishes a valuable prognostic sign. If the tension rises to normal after the inflammation has passed its acme recovery is probable, but if it falls still lower the coming destruction of the eye by atrophy is presaged, whether inflammatory symptoms persist or not. As the tension falls the eyeball shrinks, degenerative processes take place in its interior, the contraction of exudates draws the parts in various directions, so that the anterior chamber may be made deeper or shallower by the drawing backward, or the pressing forward of the lens, which becomes cataractous, and the sclera is thrown into folds, until finally the upper lid hangs down loosely over the shrunken, sightless, misshapen, very soft stump of an atrophic globe. **Phthisis bulbi** is practically the same condition produced by suppuration and evacuation of the contents of the eyeball. Sometimes calcific changes take place and areas may be felt in the ball that have a bony hardness. Such an atrophic or phthisical stump may be painless and insensitive to pressure, but often it is more or less painful, and more often tender to the touch, and it must never be forgotten that a tender, shrunken, phthisical eyeball is liable to excite sympathetic ophthalmia.

When a patient complains of a dark cloud that has appeared

suddenly, perhaps preceded by photopsia or metamorphopsia, and we find the anterior segment of the globe apparently normal, but the tension distinctly minus, we suspect a detachment of the retina. If he tells of a sudden impairment of vision, perhaps with some sensitiveness to light and more or less pain, and we find an empty or shallow anterior chamber, a small pupil, and minus tension, associated in most cases with a ciliary injection and other signs of irritation, our first thought should be of an opening in the capsule of the eye. An ulcer of the cornea, of which the patient may or may not have been aware, possibly has eaten its way through and let the aqueous escape, or the eyeball may have been wounded. In the latter case the patient is likely to be well aware that his eye has been struck, but in rare instances he is not, so we must always submit such an eye to a searching examination. In case we find a wound we must search the eye for a foreign body, for if such a body is overlooked at this time we may expect it to manifest its presence later by the reappearance of a minus tension with other signs of irritation or inflammation that usher in atrophy, with or without siderosis, though it may become encysted and do little harm.

When a careful search has revealed no opening in the capsule, no morbid changes in the eye, and the patient is not in extremis, we look for some lesion of the sympathetic nerve on the same side of the body, and shall probably find it in the cervical portion. In such cases, which are not common, the affection of the eye is apt to come and go, the patient having more or less pain and photophobia with a blurring of the vision during the attack. The upper lid droops slightly, there is a little enophthalmos, the pupil is small, the cornea may show a little wrinkling, and the tension may be as low as — 3, but no signs of inflammation or injury can be found. After a while these symptoms pass away and the eye regains its normal condition until a recurrence takes place. This is called **intermittent ophthalmomalacia.** Its prognosis is good, so far as the effects on the eye are concerned. *Roemer* states that some cases of intermittent ophthalmomalacia run a more chronic course, when the eye may become considerably smaller while circulatory anomalies appear in the affected side of the face, and that very rare cases of an apparently primary softening of the eyeball have been met with in which there were no demonstrable signs of trouble in the sympathetic nerve.

CHAPTER XIV

Injuries of the Eye, and Sympathetic Ophthalmia

When an eye has been injured we inquire into the attendant circumstances with a view to learn the nature of the object with which the damage was inflicted, and the direction from which it struck the eye, as in many cases this information is of material assistance in explaining the nature of the lesions we may find. If the object was large and blunt it produced a **contusion,** perhaps associated with lacerations, and the lids are swollen and ecchymotic in a fresh case. If it was sharp there may or may not be an **incised wound** of the lids, as well as of the eye itself. If it was a **small flying body** the lids may be closed spasmodically, but they seldom show any external evidence of a wound, whether the foreign body is or is not within the eye.

CONTUSIONS

The ecchymotic lids may be so œdematous as to be completely closed, or the patient may be able to open his eyes more or less, and the swelling may not be proportionate to the harm done. If he can see with the eye we hope to find little or no damage, but a fair amount of vision does not exclude a serious injury, while an immediate loss of sight does not necessarily imply that the eye is lost. We separate the lids with care not to press on the eye at all, and ascertain whether the cornea or sclera is ruptured or not, as a rupture is a very serious lesion which modifies our subsequent procedures.

Contusions without Rupture of the Capsule of the Eye

In the great majority of cases we find the capsule of the eyeball intact. Then we note the extent of the subconjunctival hemorrhages, observing especially whether any of them are dense enough to conceal a rupture, any abrasions that may be present on the cornea, and whether the anterior chamber contains blood. When the anterior

chamber is full of blood we are obliged to wait a few days until it has at least partially cleared up before making further observations, except with regard to the tension. Usually the blood is gathered into a hyphæma at the bottom of the anterior chamber so that we can see the larger part of the iris and pupil, and get a view into the interior of the eye.

A blow may cause little or no damage, or an immense amount of disorganization within the eye, and no criterion is furnished by the findings in any one case as to what will be found in another, so it must be understood that the lesions about to be described may be present alone in one case, in any manner of combination in another, and entirely absent in a third, even though the general nature of the contusion is the same in all three.

A **hyphæma** may come from a laceration of the iris, when the place from which the blood trickles can be seen sometimes, or from a ruptured vessel elsewhere in the eye, including the canal of Schlemm, and may or may not indicate that serious damage has been inflicted. It should disappear pretty quickly, and slow absorption is a rather bad symptom. Occasionally it blocks the outlet of the aqueous and then it causes secondary glaucoma. When the anterior chamber is filled with blood and the absorption is slow, the cornea may become stained by the imbibition of the coloring matter.

Instead of a hyphæma we may find a **gelatinous exudate** in the anterior chamber which renders the aqueous cloudy. This indicates a paralysis of the vessels of the iris and ciliary body and may give rise to secondary glaucoma.

If the **pupil is very small** and does not respond well to atropine, we look for a spasm of the sphincter, a serous saturation of the iris, and an œdema of the retina if the vision is impaired, but this traumatic myosis passes off in a few days. If the **pupil is dilated** we need to ascertain whether the mydriasis is due to a luxation of the lens, a paralysis of the accommodation, or a rupture of the sphincter. We note whether it is round or oval, the anterior chamber equally deep in all corresponding places, and the iris tremulous or not, if these details can be made out, and we test the accommodation if the vision is sufficiently good. Even though we cannot learn all of these things in every case, we can in the majority.

If the entire margin of the pupil is visible we can see whether its dark, **retinal edge** is unbroken, or if it presents minute gaps; if it is present everywhere and the pupil is round, the mydriasis probably

will be temporary, but if there are gaps in which the retinal pigment is absent the sphincter has been torn and the mydriasis will be permanent. Very rarely a gap that resembles a coloboma is to be seen when the laceration has extended into the ciliary zone of the iris, or to its peripheral margin.

Another patient may show an **iridodialysis,** and if it is quite large we may see an anteversion of the iris. In very rare instances the posterior layer alone is torn, so that we can get a red reflex, or see a cataractous lens through the iris at this place. Another rare condition is traumatic aniridia, or **irideremia,** in which the iris forms a little blackish or grayish mass at the bottom of the anterior chamber. Inversion of the iris may be met with, though not as often as when the sclera has been ruptured; this indicates a rupture of the zonule, usually associated with a dislocation of the lens.

When the vision of the eye permits us to test the **accommodation** we may find the latter paralyzed, or in a state of spasm, with a dilated or a contracted pupil, and the patient may inform us that objects appear to be larger or smaller than they do to his other eye. The prognosis is good; in uncomplicated cases the condition is not apt to last very long, but it is difficult to estimate its duration in any given case.

The **lens** may be luxated or subluxated in any direction, and may or may not be opaque. It may be placed obliquely, with one edge directed more or less forward. Sometimes our attention is called to the dislocation by the complaint of monocular diplopia, or by a change of the refraction, either to myopia or to high hypermetropia; the monocular diplopia suggests that the clear lens has been subluxated in such a way that the pupil is divided by its margin so as to cause the retina to receive two images; the myopia intimates that the lens is nearly or quite in place, but has assumed a more globular form than normal as the result of a rupture of the zonule, a phenomenon observed only in young people; while high hypermetropia tells us that the lens has been luxated completely from behind the pupil. If the patient has had from the start pain radiating through the side of the head, with a ciliary injection and an increase of tension, and if these symptoms have been increasing steadily in severity, we have good reason to suspect that the lens has been luxated forward into the anterior chamber. If the pupil is large, oval, and immobile, and the anterior chamber unevenly deep, we know that the lens is partially in the anterior chamber, and can demonstrate its

bright edge by oblique illumination. In another case the anterior chamber is uniformly very deep, and the pupil round, but by oblique illumination we may be able to detect something that resembles a drop of oil with a bright margin which has largely replaced the aqueous, and is a clear lens which is lying in its entirety in the anterior chamber. In other cases the lens can be seen more easily because it is more or less cataractous. The cornea soon becomes opaque wherever the lens lies in contact with it. According to *Foerster* a total luxation of the lens into the anterior chamber occurs only when a blow falls vertically on the center of the cornea.

When the **lenticular capsule** has been **ruptured** the lens, whether it has been dislocated or not, begins to swell and grow opaque. The rapidity of the process usually is in proportion to the size of the aperture; when the swelling is very rapid symptoms of secondary glaucoma are apt to be excited, while in other cases traumatic cataract develops more or less slowly. The process may terminate in absorption of the lens and produce aphakia without operative intervention, particularly when the patient is young, but this is not likely to happen in an adult. Once in a while an imprint of the pupil can be seen to be stamped upon the capsule of the lens. Traumatic cataract sometimes is caused without the least demonstrable injury to the capsule, and it has been proved that it can develop a long time after a contusion, even when the lens has not been dislocated, but we do not know how either of these conditions is brought about.

Several conditions may account for the fact that a patient cannot see with an eye after a contusion, although he presents no lesions in the cornea, aqueous, iris, or lens. We may be able to get no fundus reflex with the ophthalmoscope, or only a slight dark red one, but be able to see something red behind the lens by oblique illumination. In such a case a large **hemorrhage** has taken place into the vitreous, and we shall have to wait some time to learn the extent of the damage, for traumatic hemorrhages into the vitreous are absorbed slowly. The prognosis is grave, as the chances are that the retina, choroid, or ciliary body has been injured seriously, and atrophy may set in before the medium becomes clear, but it is not absolutely bad, because the blood may have come from a vessel that is comparatively unimportant, as in the following case. A boy was struck in the eye by the recoil of a broken rope. Vision was reduced to perception of light, and the vitreous was filled by a large

hemorrhage. The blood was absorbed in the course of time, the vision improved gradually until it became 20/20 with a practically perfect field, and after recovery no lesion was seen in the fundus, so that the site of the ruptured vessel remained doubtful. We must not expect such good fortune. but we welcome it when it comes.

In other cases we see flaky or striated masses of blood that move about in the vitreous, or a large red disk, either round or with a straight horizontal upper edge, between the retina and the vitreous. The presence of the latter is not necessarily an indication that the eye has been struck, as sometimes it is caused by an injury to the head, or by some other trouble which creates a disturbance in the intracranial circulation and results in engorgement, and it is the form assumed frequently by the juvenile recurrent hemorrhages of unknown origin that are occasionally met with in young men.

Hemorrhages of all sizes and shapes may be found anywhere in the retina. They are absorbed much more quickly than those into the vitreous, and differ widely in the effects they produce. They may disappear leaving little if any functional impairment, or they may set up a proliferation of connective tissue that produces the picture of a proliferating retinitis. A hemorrhage in the center of the macula is apt to leave a central scotoma that impairs the vision greatly, although a similar scotoma in the periphery might never be noticed by the patient.

A traumatic œdema of the retina is apt to be associated with a small pupil which is not affected by atropine as much as usual. An hour or two after the accident we can see whitish spots scattered about in the fundus, which increase in size and coalesce, until at the end of twenty-four hours a large part of the retina is opaque and grayish, or grayish white, inclining sometimes to yellowish. This œdema can be differentiated from a detachment of the retina by observation of the vessels, which stand out distinctly, are normal, and not tortuous in the former, but are dark and tortuous in the latter. The œdema begins to clear up at the periphery, passes away in a few days, and the patient is likely to make an excellent recovery, though often the vision is poor for a long time after all visible changes have disappeared from the fundus. In some cases a yellow or a black spot is left at the macula, and then the patient has a corresponding scotoma.

In other cases a round, red spot is found in the macula, perhaps

half as large as the papilla, through which the choroid is visible. This is known as a **hole in the macula.** Vision is lost totally in the area occupied by the hole, and the damage is irreparable.

When the media are sufficiently clear to permit an ophthalmoscopic examination we may happen to see a grayish red, hemispherical, smooth protuberance into the vitreous, over which run dark, tortuous vessels. If the protrusion is very pronounced we know that the **retina has been detached** by a hemorrhage, but if it is flat the detachment may be either of the retina or of the choroid. Very rarely a laceration of the retina can be made out, perhaps with a flap floating out into the vitreous.

In another case we observe a yellowish white stripe in the fundus, which usually is curved and concentric to the temporal side of the papilla. This is an indirect **rupture of the choroid,** and has been caused by a blow on the opposite side of the eyeball. Ordinarily there is little bleeding from the ruptured choroidal vessels. The color is yellowish rather than bluish white, because the lamina fusca remains intact as a rule. Retinal vessels may pass over the rupture, though the retina often is torn simultaneously, and when the lesion is broad the ciliary vessels sometimes are to be seen within it as red stripes. Direct ruptures, caused by the impact of a body after it has entered the orbit, are rare, and irregular in form and situation. The degree to which the vision is affected depends upon the situation of the rupture as regards the macula, and the impairment is permanent.

Secondary glaucoma may follow a contusion, and when it occurs we need to search out the cause, which is most likely to be a hemorrhage, an exudate, or a swollen or luxated lens. A possible cause which may readily escape observation is a protrusion of vitreous into the anterior chamber through a rent in the zonule. This is not common and is apt to remain unnoticed unless the failure to discover the cause of the onset of glaucoma leads us to examine the eye with a lens of high magnifying power, when we may be able to see a large, transparent drop that quivers with the movements of the eye. It is also possible for an attack of primary glaucoma to be excited by the blow in an eye that is predisposed thereto.

On the other hand the tension may be made **subnormal** by a contusion. If the eye presents in addition a marked ciliary injection, a deep anterior chamber, a hyperæmic iris, and an eyeball that is tender to the touch, we look for a vascular paralysis in the ciliary

body with œdema and hemorrhage. If these symptoms of irritation are absent we fear a detachment of the retina.

Contusions with Rupture of the Capsule of the Eye

Less often when we lift the upper lid we see a more or less jagged tear in the cornea, sclera, or both, usually with some of the tissues that belong within the eye caught between its edges. Our first duty in such a case is to clear away any detritus in the conjunctival sac, and to trim off all of the prolapsed tissues, not only as the first step in treatment, but also to enable us to observe more accurately the damage that has been done, and so gain a better opportunity to judge of the prognosis and of the best treatment to employ. This is done in the same way as when we are dealing with a penetrating wound.

The **rupture** may be large or small. A large one may pass across the entire cornea, across only a portion of it, be confined to that tissue, extend into the sclera, or lie wholly in the latter. In the last case it usually is found to run parallel to the margin of the cornea, at a distance of a few millimeters, but may vary somewhat in direction. Rarely it takes place at or behind the equator, when the lens is very apt to be extruded into Tenon's capsule. Small ruptures, as a rule, are found in the sclera alone, close to the limbus, above or internal to the margin of the cornea.

It is evident that a contusion of sufficient force to rupture the capsule of the eyeball is likely to disorganize the contents of the latter to an equal or greater degree than one which does not cause rupture, so all of the lesions just described may be found in these cases also. In addition, more or less of the contents of the eyeball are extruded, as a rule, and a place of entrance is opened to infection, so the prognosis is worse. Yet the condition is not hopeless, for ruptured eyes have been saved with useful vision. *Roemer* tells of a patient both of whose eyes were ruptured with extrusion of the lens in two successive years by the horn of a cow, yet they recovered with good vision. Such an experience is phenomenal, but an eye must not be despaired of until it is demonstrated to have suffered a vital injury.

The black tissue snipped away from between the lips of the wound may have been **iris** or ciliary body. If we have been obliged to cut away a part of the latter the probability is that the eye is injured

fatally, and we must consider that the danger of sympathetic ophthalmia has been greatly increased.

The entire iris may have been torn away and extruded from the eye, usually in association with the lens, though the latter has been left in place in rare cases. We may find it in the conjunctival sac, beneath the bulbar conjunctiva, or caught in the rupture. Otherwise the lesions of the iris are about the same as in contusions without rupture, except that inversion is more common.

The **lens** may remain in place, but it is either subluxated or luxated in most cases, and often it is extruded, when it may be caught in the wound, be found beneath the bulbar conjunctiva, sometimes at quite a distance from the rupture, or in the conjunctival sac. Occasionally when the conjunctiva is not torn, a rounded prominence surrounded by a mass of blood can be seen near the cornea; the probability is that such a prominence has been made by the lens, which has been forced through a rupture of the sclera, that very likely it is covered and hidden by the lens and blood.

Vitreous has escaped, as a rule, leaving the eyeball more or less collapsed immediately after the accident. If the tension becomes normal again after the wound has closed, we have some hope of saving the eye because enough fluid has been secreted to compensate for the loss, but if this does not happen the prognosis is bad. The sight is not likely to be very good when an eye is saved after such an injury, for it often is left more or less deformed and very astigmatic.

PENETRATING WOUNDS OF THE EYE

A wound made by the penetration of some object into the eye is far more common than a rupture of the capsule from contusion. The history of the case generally informs us if the eye has been punctured by a knife, a pair of scissors, an arrow, a splinter of wood, a pointed wire, a nail, or some similar object, and the instrument itself often is presented for inspection, or if it has been struck by some flying missile, like a chip of steel, glass, stone, copper, or perhaps a small shot.

After taking the history we separate the lids carefully and find a wound in the cornea, or in any part of the sclera that could be exposed, which corresponds in size and shape to the instrument with which it was made. If it is in the cornea we are likely to find the

anterior chamber shallow or abolished, and a prolapse of iris, but we can perceive the wound easily, even when there is no prolapse to mark its situation, because its margins are swollen and slightly gray. The iris must be trimmed away because it interferes with our inspection and will invite trouble in the future if we leave it alone. To do this we cleanse the eye, apply an anæsthetic, seize the prolapse with forceps, draw it out a little so as to break up any adhesions that may have been formed, and cut it off with scissors close to the surface of the cornea. What is left of the iris will drop back into place, and then we can follow the track of the wound more clearly to ascertain what further damage has been done.

It is possible for the cornea to be struck obliquely by a narrow instrument, like a sharp pointed crochet needle, so as to make a wound which closes like a valve and allows only a small quantity of the aqueous to escape. The anterior chamber then may seem to be of its normal depth, but the tension is subnormal immediately after the accident. On one occasion I had the opportunity to see what looked like a particolored ribbon lying in the cornea after an accident of this nature. A blunt spud introduced into the wound relieved the pressure of its lips upon the bit of iris, which withdrew suddenly and sprang back into place. The wounding instrument had passed obliquely through the cornea and stopped; when it was withdrawn a gush of aqueous had carried a bit of the margin of the iris along and had stretched it out in the wound, where it was held tightly until the valvelike closure was relieved.

While we are dealing with the prolapse of iris we notice whether it contains any lens matter or vitreous, for if either of these are present the lens or the zonule has been wounded. Then we observe whether the lens is clear, or is becoming swollen and opaque, whether there is any blood in the anterior chamber or vitreous, and satisfy ourselves as well as we can what tissues have suffered.

Foreign Bodies in the Eye

The next point to be decided is most important—whether there is a foreign body in the eye. This is highly improbable in many cases of stab wound, though it is possible for an **eyelash** to have been carried into the anterior chamber or iris, and if this happens trouble is likely to be caused later; but this danger is so remote that we seldom do more than look as carefully as we can for such a lash

without disturbing the eye unduly, and proceed to look for symptoms of infection, and then to begin treatment.

If the wound has been made by a **small flying missile,** or by a substance that can leave particles behind, like a splinter of wood, a thorough search must be made. Sometimes a foreign body can be seen in the anterior chamber, entangled in the iris, or sticking in the lens, or an abnormal lump in the iris may suggest its presence behind the last. Perhaps an aperture in the iris shows the course it has taken. After the pupil has been dilated we can sometimes see it in the vitreous, or somewhere in the fundus; more rarely we can detect a second wound in the posterior part of the globe through which it has escaped, or a little white spot with edges tinged with blood that marks a place where it cut the retina and choroid and rebounded. If it has been in the eye for several weeks it is possible that we may see a white spot surrounded by pigment in the retina, which marks where it lies embedded in exudate. But in the great majority of cases the foreign body is hidden by the normal structures of the eye, by blood, by exudates, or by an opacity of the lens, so that we cannot locate it by the power of vision. If it happens to be composed of a magnetic substance we may be able to detect its presence by means of a sideroscope, or of a magnet, but the diagnostic value of these instruments has been cast in the shade by the modern development of radiography.

Although the great majority of the patients are well aware when an eye has been struck by a foreign body, ***it is possible for a small missile to enter without being felt*** and to cause none but very transient symptoms. In such a case the patient may notice that his vision is blurred, and we may find the anterior chamber shallow, the pupil small, and the tension minus, symptoms which guide us to look for a wound in the capsule, and then for a foreign body within the eye; otherwise he is apt to come under observation later with commencing atrophy of the eyeball, with or without siderosis. Rarely the wound heals, the tension and the vision return to normal, and the foreign body becomes encapsulated, later perhaps to become a puzzle to the examiner. A boy, 14 years old, was brought by his parents to be examined for glasses. A round, greenish object, about the size of a birdshot apparently, could be seen in the fundus. The boy denied that his eye had ever been hurt, his parents claimed that the eye had never been sore. The nature of the object in the fundus was a puzzle to a large number of skilled ophthalmologists at the

New York Academy of Medicine until the late *Dr. Herman Knapp* suggested the probable diagnosis. A very careful inspection revealed a scar in the cornea, a dehiscence in the iris, and a white track bordered by pigment in the choroid, all forming a line that led directly to the round, green object. The patient has now been under observation for nearly twenty years, the conditions have not changed, and the most probable diagnosis seems to be that a birdshot entered his eye without causing pain or any other trouble and now lies encysted in the choroid.

The **X-rays** will not detect fragments of glass or wood, or any object less than one millimeter in diameter, yet they furnish the best means now at our disposal for the detection of intraocular foreign bodies. An ordinary radiograph informs us simply that a body impervious to the rays is in the orbit, but its location therein can be determined definitely by means of the apparatus invented by *Mackenzie Davidson* and described in the Transactions of the Ninth International Ophthalmological Congress, 1899, or better perhaps by the one devised by *W. M. Sweet.* The latter has kindly furnished me the following brief account of his apparatus:—

"In the earlier form of this apparatus a plate holding device was attached to the side of the head of the injured eye, and two ball-pointed rods connected with the holder were adjusted until one of the balls was opposite the center of the cornea. Two negatives were made, which gave a different relation of the foreign body in the eyeball or orbit to the two indicating balls, and by means of measurements of the plane of shadows at the two exposures, the position of the steel was plotted on a localizing chart. In a more recent form of the apparatus only one ball is employed, as the tube holder, indicating the ball, and plate holder are upon a movable stage, and therefore present a known relation to each other which does not vary. The two exposures are made upon one plate. Since the relative positions of the tube to the indicating ball and photographic plate remain fixed, the direction of the rays in passing through the eyeball always follows a definite course. The localizing chart has therefore been ruled with lines to indicate the rays from the tube to the plate. By placing the developed plate in a keyplate of focal coordinates it is possible to determine the situation of the foreign body by reading from this keyplate and transcribing the readings to the localizing chart."

No instrument should ever be introduced into a wounded eye in

search of a foreign body that has not been located, no matter how conclusive the evidence may be that one has entered. Such a method of search may do irreparable harm to an eye capable of recovery from the injury, for the missile may have ricocheted. Before radiography was commonly practiced a child was struck in the eye by a flying particle from a cold chisel; the eye was wounded, the media clear, no foreign body could be seen, and no dehiscence was detected in the iris, but the child screamed with pain when the electric magnet was brought near, and stopped as soon as the current was cut off from the instrument, which seemed pretty good evidence of the presence of a magnetic foreign body within the eye. Finally the upper lid was noticed to bulge when the magnet was brought near the wound, and a fragment of steel was found entangled in the upper transitional fold of the conjunctiva, to which place it had been deflected after it had cut the cornea. Such a case as this is uncommon, though it is not unique, for under similar conditions shot have been found in the conjunctival sac, and we must always refrain from any measure that is likely to do harm rather than good.

Three great dangers attend every penetrating wound of the eye, aside from those attendant upon the injuries inflicted directly and the presence of a foreign body:—1, infection and suppuration; 2, inflammatory changes; 3, sympathetic ophthalmia.

INFECTION OF A WOUNDED EYE

Septic material may be carried into the eye by the penetrating substance, or come in contact with the wound subsequently. We cannot always be sure which has happened if the symptoms appear in the wound within two or three days, but we know that the infection took place at, or shortly after the time of the accident, while if they appear at a later date than this we realize that our precautions to prevent infection have not been sufficient.

If the wound is in the cornea the **first sign** of infection is likely to be a slight cloudiness of the surrounding tissue. A few hours later we find that this cloudiness has increased, that the gray margins of the wound have become yellowish, and then pus appears in the anterior chamber. When the wound is in the sclera it is not probable that we shall see the infiltration of its margins, and the first signs that we notice may be those of an onset of iritis, or the appear-

ance of pus in the anterior chamber. We should never fail to make a bacteriological examination of the matter from the margin of the wound, that we may learn with what organism we have to deal. Less frequently the margins of the wound are not infected, but septic material has been carried deeply into the eye. Intraocular infection presents several different pictures according to its extent and the tissues that are involved. The worst form is the one called panophthalmitis, in which all of the tissues are involved, and this only too often proves to be the final stage of the others.

Panophthalmitis

When a patient develops a considerable degree of fever and of pain in the wounded eye, the lids of which rapidly become very œdematous and closed over a protuberant and immovable eyeball, while the conjunctiva is chemotic, the margins of the wound are of a dirty yellow, the aqueous is turbid with a hypopyon, the iris is inflamed, and perhaps the lens is swollen and opaque, a virulent infection has induced suppuration of all the tissues within the sclera. The prognosis is bad.

Infection of the Anterior Segment of the Eye

Sometimes the patient does not have so much fever and pain, no exophthalmos, and much less œdema of the lids and chemosis, although the margins of the wound are yellowish, there is a fibrinopurulent exudate in the anterior chamber, and the picture of a purulent iritis is presented. In such a case the suppuration is confined to the anterior segment, and there is a chance that we may save the eye, though the prognosis is not good. When the ciliary body is involved, the severity of the symptoms is intermediate between that seen when the tissues involved are more anterior and that seen in panophthalmitis, and the lens may be enveloped in a mass of exudate. When an inflammation of this nature starts primarily in the iris or ciliary body, we know that the infecting material was carried directly to it by the foreign body. In other cases the color of the wounded, swollen lens is yellow instead of gray, showing the presence of suppuration within; prompt evacuation of the purulent mass saves such an eye occasionally, but not often.

Abscess of the Vitreous

A wounded eye may not seem to be irritated any more than we should expect it to be if the wound were aseptic. Whether there is a prolapse of iris or not we may find little or no swelling of the lids, or chemosis of the conjunctiva, no more than a moderate ciliary injection, no unusual pain or tenderness, in short no external or subjective symptoms which would lead us to suspect the eye to be in a desperate condition. Yet, when we cast light into the eye by oblique illumination, if we see a greenish yellow reflex from deep in the vitreous we are made aware that septic material was carried into the eye by the wounding instrument, and that an abscess has formed in the vitreous. If the amount of pus is small we may obtain an indistinct red reflex with the ophthalmoscope, but this reflex is blotted out as the pus increases in quantity.

The prognosis is very grave. Panophthalmitis develops in a few days, as a rule, though once in a while the abscess becomes encapsuled, the pus absorbed and replaced by connective tissue, but this result is nearly as fatal to the eye. The shrinkage of the mass usually detaches the retina, sometimes the choroid, and finally the eye is lost through phthisis bulbi. It is with extreme rarity that an eye survives an abscess of the vitreous with the preservation of any vision.

INFLAMMATORY CHANGES INDUCED BY WOUNDS

A proliferation of new connective tissue may be set up in the hemorrhages and exudates that follow a wound of the eye, even when it is aseptic, and the contraction of this tissue may cause intense pain by the tension exerted on parts supplied by sensory nerves, detach the retina, or induce atrophy of the globe through its interference with nutrition, or it may simply impair the sight more or less, all depending on its situation and extent.

An iridocyclitis that persists after the wound has healed is a very serious condition, no matter how slight it may be, or whether the eye is functionally capable or not. A zone some 5 mm. broad about the eye, its anterior edge somewhat back from the margin of the cornea, is called the **danger zone of the sclera,** because wounds in this region seem to be more prone than others to be followed by an iridocyclitis that gives rise to sympathetic ophthalmia. We view a

wound in this region with apprehension. It may affect the sclera and nothing else, when it will heal smoothly if it is aseptic, and leave the eye perfectly well. But if the ciliary body has been wounded we should consider thoughtfully the amount of other damage that has been inflicted before we decide whether we are justified or not in an attempt to save the eye. If the other damage is sufficiently slight to warrant the hope that the eye will retain or regain good vision, we should make the attempt after we have informed the patient of the risk, for the expected iridocyclitis does not always develop. If the decision is to enucleate and the patient declines the operation, it is best to have the statement that he assumes the responsibility made in writing, or at least before several reliable witnesses, as a protection against a suit for damages should he subsequently lose the other eye through sympathetic ophthalmia.

An iridocyclitis capable of exciting sympathetic inflammation in the other eye may be inaugurated by any lesion that opens the capsule of the globe, like an incision for a cataract extraction, or the perforation of an ulcer of the cornea, when the ciliary body is not wounded, but the proportionate number of cases in which sympathetic trouble develops seems to be much smaller when the ciliary body has been spared.

The iridocyclitis may be of any degree of severity, its symptoms slight or well marked. After the wound has healed there may be only a little tenderness over the ciliary region, and a tendency on the part of the eye to be irritable, to become injected, to lacrimate, and to show some photophobia on slight provocation. Yet this chronic, low grade inflammation is apt to prove very destructive, and the danger of sympathetic ophthalmia from it is quite as great as, if not greater than, when the wound is followed by a severe plastic iridocyclitis characterized by intense pain in the side of the head, fever, vomiting, tenderness of the eyeball, œdema of the lids, chemosis, a marked ciliary injection, and exudates behind the iris, into the pupil, into the vitreous, and into the ciliary body itself.

SYMPATHETIC OPHTHALMIA

Symptoms and lesions that originate from an injury or disease in one eye but appear in the other are called sympathetic. If the sympathizing eye is inflamed the condition is one of **sympathetic inflammation,** if it is not inflamed we speak of **sympathetic irri-**

tation. We need to differentiate these conditions because sympathetic irritation appears in connection with many lesions that do not give rise to sympathetic inflammation, and is not of itself destructive. At the same time the two conditions often coexist, and the irritation may seem to be the precursor of, or to usher in the inflammation, for which reason they frequently have been confounded. It is important that we should be acquainted with the distinctive symptoms of each, the conditions under which each may be met with, and the vital difference that exists between them, in order to avoid a serious error in diagnosis.

Sympathetic Irritation

A patient with a foreign body on his cornea, or beneath his upper lid, may have his other eye reddened, watery, perhaps a little photophobic, or feeling as if it also was afflicted in the same way. These symptoms disappear after the foreign body has been removed, and we say that the eye was irritated sympathetically, but no matter how long the foreign body might remain, no inflammation would develop in the sympathizing eye in consequence. Other patients with diverse injuries or diseases of one eye, some trivial, some very serious and including wounds, exhibit this irritation in various ways. One may have a variable degree of trigeminal neuralgia about the other eye. Another may not be able to do near work for any length of time because his eye tires easily, showing that his accommodative power has been weakened, and occasionally his eyeball may be a little tender over the ciliary body. Another may complain of photopsia, or of muscæ volitantes, or that he cannot see as well as usual. In the last case we may find the vision reduced and the field contracted, with no sign of inflammation within the eye, beyond at most a hyperæmia of the fundus, or perhaps a slight haziness of the papilla. In rare cases a spasm of the accommodation with myosis has been observed. Each of the symptoms mentioned may occur alone, or in combination with one or more of the others, and all can be explained as due to reflex irritation of the ocular nerves by the lesion in the other eye. They may appear after enucleation, or any of the substitutes for that operation, may be caused by the calcification of an uninflamed stump, or by the wearing of an artificial eye, but they pass away with the removal of the exciting cause, and do not of themselves indicate the presence of inflammation, or threaten the integrity of the eye.

The interval between the occurrence of the lesion in the one eye and the onset of symptoms of sympathetic irritation in the other varies from a few seconds to a very long time; it is said to have been many years in duration. When the irritation appears quickly after a traumatism, or at any time after one in which the capsule of the eye has not been broken, or in disease that is not of traumatic origin, we may view it with more or less tranquillity, but when its onset is more than a week after a wound it is a matter of grave concern, for then it may portend an attack of sympathetic inflammation, and we must watch carefully for definite signs of iridocyclitis, or neuroretinitis.

Sympathetic Inflammation

In a typical case of sympathetic inflammation a patient who has received a penetrating wound of one eye develops iridocyclitis in the other after an interval of from one to three months. The question whether this inflammation in the second eye originated from the wounded one, or from some other cause, can be answered with a high degree of probability, but not with absolute certainty in any case. The first prerequisite for the occurrence of sympathetic inflammation is that **iridocyclitis attacked the injured eye,** and it is believed generally that the capsule must have been opened. The opening may have been made intentionally, as in an operation, or by the perforation of an ulcer, but, as a rule, it was made accidentally, and in most cases the ciliary body was wounded. Usually the wounded eyeball is more or less atrophic, but occasionally it is in good functional condition, exhibiting only a little tenderness in the ciliary region and a slight degree of irritability.

Various writers have reported cases in which they claimed that sympathetic ophthalmia originated from such troubles as contusions without rupture of the capsule of the eye, symblepharon, tattooing of the cornea, calcification of the choroid and ciliary body, cicatricial pressure on the optic nerve after enucleation, and the wearing of an artificial eye, but most if not all of these claims were made before anything like a clear distinction had been made between sympathetic irritation and sympathetic inflammation, and the evidence frequently adduced, that the symptoms disappeared after the removal of the exciting cause, would be accepted today in favor of the supposition that the condition was one of irritation. Still we cannot rule with finality that the capsule of the eyeball must be opened to per-

mit symptoms of sympathetic inflammation to be excited, for several recent writers have maintained, on the basis of studies of enucleated eyes, that an intraocular tumor which has not ruptured the capsule is able to excite an iridocyclitis that may give rise in turn to a sympathetic iridocyclitis. The pathological appearances may be misleading in this, and I have not been able to find a definite case in which sympathetic inflammation was actually excited in this manner, but we can only say as yet that in the overwhelming majority of cases sympathetic inflammation occurs only after a wound of the other eye.

Having learned that the first eye was wounded, that an inflammation of the uvea followed, which was plastic, as a rule, but may have varied a great deal in severity and just possibly may have been purulent, that the eyeball is tender to the touch at the present time, and probably that its tension is minus, we note the nature of the inflammation in the second eye, for every form except uveitis and neuroretinitis can be ruled out at once as not sympathetic. At least, as a dogmatic assertion that inflammations of other tissues of the second eye, which may occur after the first has been wounded, can never be of sympathetic origin, is out of place in view of the facts that the nature of sympathetic inflammation is still a matter of discussion, and that our diagnosis of it is one of probability rather than of certainty, we can safely say that it is extremely doubtful if they are ever connected in any way with the trouble in the wounded eye.

Sympathetic inflammation takes the form of an **iridocyclitis** in the great majority of cases. It may start in with, or be preceded by symptoms of sympathetic irritation, or it may begin as a serous cyclitis, and then the first intimation the patient receives of his condition may be dimness of vision. In such a case we may be able to see at first only a slight ciliary injection, a cloudiness of the aqueous, and a collection of little gray, or brown spots on Descemet's membrane, but other symptoms soon appear. Such a gentle attack leads us to hope for a mild course and a possible recovery, but the prognosis is grave. The entire uvea is inflamed, the vitreous will become cloudy, and the exudate is prone to become more fibrinous.

The symptoms are apt to be those of a plastic iridocyclitis from the start. Posterior synechiæ are formed, which may be broken up at first by atropine, but new ones appear continually, the pupil grows smaller steadily in spite of the drug, becomes occluded and excluded, the ciliary zone of the iris becomes bulged forward by the

aqueous accumulated in the posterior chamber, so as to form a crater pupil, and secondary glaucoma develops. In still worse cases a total posterior synechia is formed, the iris looks thickened and presents radiating folds. In the early stage of this disease we may be able to see large opacities in the vitreous, but later these are hidden by the occlusion of the pupil. In the course of weeks or months, during which there may occur periods of improvement, a complicated cataract appears, the cornea becomes opaque from glaucomatous tension, the exudates begin to shrink, the eyeball to soften, and to pass into atrophy. Quite rarely a very malignant case sets in with chemosis and severe cyclitic pain, but ordinarily the pain is not very bad until the onset of the glaucoma. The course is slow and obstinate in all cases, whether they are mild or severe. Occasionally when the patient complains of dimness of vision we may find a typical **neuroretinitis,** or possibly a commencing postneuritic optic atrophy, sometimes alone, more often associated with a uveitis. When alone its course usually may be checked by the enucleation of the wounded eye, and the latter was found to be the seat of an intense neuroretinitis in a case reported by *Pooley*. Cases of sympathetic optic atrophy have been recorded, but it is quite possible that in these the atrophy resulted from a previous neuroretinitis.

The **interval** between the wound of the one eye and the onset of sympathetic inflammation in the other usually is between one and three months, but may be shorter or longer. Some writers give the shortest known interval as seven days, others as fourteen when the signs of sympathetic irritation are not taken into account, while the longest is perhaps thirty-nine years. The longer the interval the more questionable the diagnosis must be, as there is nothing characteristic in the appearance of a sympathetically inflamed eye, and the same causes can excite an ordinary iridocyclitis or neuroretinitis when the other eye has been wounded as when it is intact. This doubt is strengthened when the wounded eye, or its stump, is neither tender, irritable, nor inflamed. At the same time we know that a phthisical stump may remain quiet for an indefinite period and then suddenly become tender, inflamed, and competent to excite sympathetic inflammation many years after the injury was inflicted, that sympathetic irritation sometimes is quite difficult to differentiate from inflammation in such a case, and that the removal of the stump has an ameliorating influence.

The only **prophylaxis** against sympathetic inflammation is the

immediate enucleation of the wounded eye, but this is not always justifiable. We seldom think of doing such an operation when the eye has good vision, unless the ciliary body has been wounded, and then we are likely to wait until an iridocyclitis has made it almost certain that the eye will be lost soon through phthisis, and that instead of saving the wounded eye we are taking a great chance of losing both eyes. This seems to be the right course to pursue, but it is also right that the patient whose ciliary body has been wounded should be informed of the risk he necessarily runs when we make an attempt to save the eye. Statistics dealing with the frequency of sympathetic ophthalmia in this class of cases seem to be wanting, so we cannot give him any idea of the degree of risk, except one based on personal experience, which is apt to be unreliable. On the other hand, a wounded eyeball that has become blind and phthisical as the result of an iridocyclitis should be enucleated just as soon as the patient will give consent. Enucleation may not prove a sure preventive, as sympathetic ophthalmia is said to have been known to develop after it had been performed, but such an experience is exceptional. No substitute operation is equally efficient.

A number of ophthalmic surgeons at the front in the war going on at the present time in Europe have remarked the rarity with which sympathetic inflammation has been observed to follow wounds of the eye by the missiles of warfare. The reason for such an immunity, if it is finally proved to exist, is unknown, but it has long been recognized that there is a class of cases in which wounded eyes are rendered blind, but do not suffer from iridocyclitis or atrophy, and that they do not seem likely to excite sympathetic inflammation.

CHAPTER XV

A General Consideration of the Fundus

When we come to deal with the fundus we are called upon to study the details of a picture presented as it were at the back of a room into which we can see only through a keyhole. We cannot see the whole of the picture at any one time, but must carry in our minds what we have seen in one part while we are looking at another, and join all the parts together mentally to form a whole. Each picture varies from every other in certain details, just as faces differ in their features, and sometimes there is nothing in a detail itself to tell us whether what we see is a physiological or a pathological deviation. Gross departures from the normal are perceived readily to be such, yet it is not always easy to determine their nature or even their exact site, regarding which our judgment has to be guided by a consideration of all of the details presented, together with the history and the effects which are produced on the vision and the fields for white and colors, while minute variations furnish opportunities for the judgments of experts to differ widely. Before we attempt to solve the puzzles furnished by pictures of the abnormal fundus it is essential that we should become familiar with those presented by the normal, at least in their main outlines, that we may recognize the principal physiological variations, and gradually learn by experience to differentiate minor ones from pathological changes.

The picture of the fundus is composite, formed by the effects produced by light reflected from the retina, choroid, and a circular or ellipsoidal area of a bright pinkish color that marks the termination of the optic nerve within the eye, which is called indifferently the papilla, the optic disk or disk, and the head of the optic nerve, and is the most conspicuous feature normally. A system of vessels emerges from about the center of the papilla to ramify over the rest of the fundus, and form, after they leave it, the only part of the retina that can be seen as such, although the pigment epithelium makes its presence or absence manifest through modifications of the background, which depend upon the abundance or the scarcity of the

pigment it contains. The choroid can be seen only when this pigment is so scanty as to allow us to see through the retina.

THE PAPILLA

The **size** of the papilla seems to vary with the refraction of the eye, so that its upright image appears to be larger in myopia than in hypermetropia, but in reality it varies very little, and therefore its diameter forms a convenient unit of measurement for other objects, as well as a center from which we are able to compute distances, and to indicate directions. The diameter of the papilla is 1.5 mm., and we may say that a certain lesion is one half of a papillary diameter, that is three fourths of a millimeter across, and that it is located 1½ p. d., or 2.25 mm., from the lower border of the optic disk. The papilla is round, though commonly it seems to be ellipsoidal with its long axis vertical, less often horizontal or oblique. This elongated appearance is to be ascribed in most cases to astigmatism, but it is occasionally due to a foreshortening.

The **color** of the papilla has been likened to that of a peach blossom, and is darker on the nasal than on the temporal side. It can hardly be described accurately, for it varies with the number of the red rays in the artificial light we happen to use, and we shall find the difference quite noticeable if we examine it first with either a gas or an electric light and then with the other. The true color of the papilla, as well as that of the entire fundus, can be seen only when we employ daylight, but as the use of this is impracticable we become habituated to artificial light and learn by experience to recognize the color to be or not to be normal. The fundamental tone is a creamy white, due to the reflection of the light from the white lamina cribrosa through the translucent nerve fibers, in which the yellow becomes more pronounced with the advance of age. This creamy white receives a delicate pinkish tone from the blood in the deep capillaries, and is overlaid by a shade of pink or red, the depth of which depends on the number of the superficial capillaries, the density of the nerve fibers, and perhaps on the presence of connective tissue. The general effect is influenced by the degree of pigmentation of the rest of the fundus, and by the age of the patient, as it is redder in young people and yellower in the old, yet we should study it with care, for it is affected in pathological conditions, and a slight abnormality in color often is a diagnostic point. After we

have had considerable experience we learn to distinguish a certain healthy tone.

A distinctly white, gray, or blue color, or a great redness of the papilla is perceived readily and recognized to be a pathological symptom, but slight degrees of pallor or redness may be of pathological origin. Sometimes these slight variations can be detected by comparing the two papillæ, which usually are alike in color when they are normal, and with experience we gain an uncertain ability to detect them otherwise, probably from the lack of a healthy tone, but this seldom amounts to much more than the creation of a vague suspicion that something is wrong, and this suspicion must be confirmed or disproved by the presence or absence of other symptoms.

The Margin of the Papilla

The entire margin of the papilla should be visible, except where it is covered by the blood vessels, and should be perfectly distinct, yet not too sharply defined. This is a distinction that is hard to make on paper, though it can be appreciated quickly when we compare the margin of a normal disk with that of one which has undergone simple atrophy; in the former we see it clearly through the transparent nerve fibers, but in the latter it stands out with an added degree of sharpness. The temporal margin is more distinct than the nasal, but any obscuration of any portion of the margin must be due to one of three things, a refractive error, a pathological condition of the nerve head itself or of the immediately adjacent retina, or a projection of the sclera, choroid, and retina over the nasal margin, called by some a **supertraction,** which is met with only in the conus and posterior staphyloma of high myopia. In a case of supertraction the covered part of the papilla is apt to appear like a yellowish crescent, and this has been called by *Adam* the **supertraction crescent.**

In some cases the margin of the papilla can be resolved into two rings, a white within a black. The white ring may be caused by the edge of sclera shining through the overlying transparent tissue, or by a formation of connective tissue, we cannot tell which in any individual case, so it is called either the **scleral,** or the **connective tissue ring.** The black or **pigment ring** is produced by a thickening of the retinal pigment. Both of these are perfectly normal conditions. It seldom happens that both of these rings are present and

complete, in most cases we see segments of them, at least on the temporal side of the papilla, but either one or both may be absent, or the black one may be replaced by an accumulation of black pigment spots that are apt to suggest a pathological origin when this is not excluded by the absence of other signs of disease.

The Physiological Excavation of the Papilla

In the majority of cases we see a white or whitish spot either in the center of the papilla, or encroaching on its temporal side, which marks the site of a physiological excavation. Sometimes it approaches closely to the temporal margin and adds to the pallor of this part of the disk, but if we observe accurately we shall almost if not quite invariably find a narrow zone of pinkish tissue between it and the margin. The center of the spot may be perfectly white, or it may be speckled with gray dots that show the meshes of the lamina cribrosa, but if it is not very deep it is more likely to have a pinkish tone which is much lighter than that of the surrounding tissues, and grows darker toward the periphery in many cases.

The physiological excavation is a depression formed by the separation of the optic nerve fibers which rise through the aperture in the sclera and choroid and then bend in various directions to be distributed throughout the retina. If these fibers fill the aperture completely they separate on a level with the retina, form almost no depression, and the white spot in the center of the papilla is nearly or quite absent. We speak of such a papilla as flat because it presents no excavation. In most cases the fibers do not quite fill the aperture, and as they draw apart they leave a depression which may be large or small, shallow or deep, and have gently sloping, or precipitous walls. The size of this physiological excavation, or cup, is indicated by that of the central white spot. It may be simply a depression in the surface of the papilla, or a hollow that reaches down to the lamina cribrosa. A small one may be deep, a large one shallow, or the reverse. If blood vessels pass over its edge and continue their courses unbrokenly, its walls form a gradual incline; if the courses are almost direct and the vessels remain distinct to the place where they plunge down into the tissue of the nerve, the excavation is shallow; if they curve after they have crossed the edge and grow rather indistinct as they approach the center, or if gray dots are to be seen in the floor of the cup, it is deep. When the

vessels hook over the edge, become very dark, lose their light streaks, and become very indistinct or disappear, the walls of the cup are precipitous or abrupt. If apparently disconnected, indistinct vessels are to be seen in its floor, we know that the excavation is large, deep, and has precipitous sides.

Differentiation of Physiological from Pathological Excavations

When the papilla is of a normal color, and the white spot that marks the site of the excavation is surrounded by a pinkish zone, the cup is **physiological,** no matter whether it is large or small, shallow or deep, and has abrupt or gently sloping walls. When the disk is abnormally white, gray, or blue, an excavation with gently sloping walls that reaches its margin is a sign of atrophy, and is called **atrophic,** though probably it is little if anything more than a large physiological excavation that has been accentuated by the atrophy of the nerve fibers. A cup with precipitous walls that reaches to the margin of a similarly discolored papilla of normal size is an infallible sign of glaucoma. The differentiation of these three varieties is easy in well marked cases, but when the conditions are not well defined it is apt to be difficult. When the papilla has a good color, while the excavation touches, or seems to touch its margin, the differentiation of a physiological from a glaucomatous cup depends on whether its wall is gently sloping or abrupt at the place of contact, and this is determined by observation of the courses of the vessels that cross the margin. If they pass over without bending we know that the cup is physiological, but if they hook over and seem to form little dark knobs as they pass, it is **glaucomatous.** The presence of a glaucomatous halo may prove a bit confusing when we have to look sharply to distinguish it from the nerve tissue by its color, on account of the absence of the scleral and pigment rings, but with care we can usually see that the color of the halo and that of the discolored nerve tissue is not exactly the same. We seldom need to go to this trouble, however, because the halo is a late symptom in glaucoma, and when it is present we have other symptoms on which we can rely for our diagnosis.

An extreme rarity in which a large excavation with precipitous walls may be in contact with the margin of the papilla and yet not indicate glaucoma, is the **coloboma of the optic nerve sheath.** The papilla is two, three or four times as large as normal, and

glaucoma may be excluded by the absence of other symptoms of this disease, as well as by the nonprogressive character of the lesion.

The Vessels of the Papilla and Retina

The pictures presented by these vessels vary a great deal, largely because minute differences in the sites of their branching produce marked effects on the appearance. The central artery and vein divide each into two branches, one of which runs upward, the other downward, to form the superior and inferior artery and vein. Usually this bifurcation takes place just before, or as the central vessels reach the surface, when we see one artery running up, and another running down on the surface of the disk, each accompanied by a vein, from a place in the physiological excavation about the center of the nerve head, where they are separated by a small interspace, or seem to fuse. Each of these vessels divides into two branches, the superior temporal and nasal, and the inferior temporal and nasal arteries and veins, either before or after it has left the disk to enter the retina. These divide and subdivide into continually smaller branches that spread through the retina, but the subdivision is by no means regular and symmetrical. Less often the central vessels divide farther back in the nerve, when the superior and inferior branches emerge from the nerve tissue separated by a much wider interval, or we may see three or four arterial and venous trunks come to the surface in widely separated parts of the nerve head, when a subdivision of these branches has already taken place. In still other cases short portions of the central vessels are visible before they divide.

Each artery is accompanied by a vein, but the association does not seem to be very close among the smaller vessels. On the papilla the arteries are placed on the nasal side of the veins, where they are distinguished readily, as they are slender, sharply outlined, of a bright red color, and along the middle have light streaks which are about a quarter as wide as the vessels themselves, while the veins are broader, darker, and have much narrower and less distinct light streaks. These differences become less evident as the vessels grow smaller, so that sometimes we have to trace a small vessel until it joins a larger one in order to determine whether it is an artery or a vein. Arteries and veins cross each other frequently, sometimes with one, sometimes with the other in front, but ***an artery never crosses an artery, and a***

vein never crosses a vein; if an exception to this rule exists it must be one that is very rare. Both arteries and veins tend to be tortuous, the veins the more so, as a rule. At times the large vessels may be seen to be twisted about each other, the artery taking its natural course and the vein winding about it in most cases, or one of them may form a twisted loop, which occasionally projects into the vitreous, where it is liable to be mistaken for the remains of a hyaloid artery. The **tortuosity** of the vessels is much greater in some persons than in others, and as tortuosity often is a pathological symptom we need to know when it is physiological. It is only in rare cases that a physiological tortuosity is not present and of a similar character in both eyes. A physiological bend is always in the same plane as the retina, with the one rare exception of a twisted cord projecting into the vitreous from the papilla. Hence we compare the tortuous vessel with the corresponding one in the other eye, and note the plane in which the bend takes place. Any anteroposterior curvature, except at the point where the vessels turn to plunge into the nerve, is to be looked upon as a pathological symptom.

The vessels are largest where they bend to enter the nerve, but occasionally they appear to taper at this place. In the case of an artery we refer this appearance to a foreshortening caused by an oblique course, or to the presence of a certain amount of connective tissue, but if a vein seems to taper at this point we think at once of an increased intraocular tension.

We do not see the vessels themselves because their **walls** are transparent, we see only the blood columns that they contain. The one exception to this statement, as regards the normal eye, is that occasionally we can make out very narrow, pale yellow, or whitish lines along the sides of the blood columns on the papilla, rarely after they have entered the retina. All other opacities, except medullated nerve fibers, along or over the vessels are of pathological origin. In young people we frequently see a bright border along the side of a vessel, but when we move the mirror a little it changes its place or shape, or disappears, thus proving itself to be nothing more than a light reflex. The outline of each blood column should be smooth and even; any irregularity is to be ascribed to some fault in its wall and is to be looked upon with apprehension. The light streak, which is of uncertain origin, should be uniform, and any irregularity leads us to suspect trouble in the vessel itself.

The **size** of the retinal vessels varies in different persons, so that

slight abnormalities in this respect are very difficult to detect. We study the relative diameters of the arteries and veins, which should be about as three is to four, and compare corresponding vessels in the two eyes, in which they commonly are alike, and as experience increases we become able to detect slighter and slighter variations from the normal. Any marked deviation from the proportionate sizes of the arteries and veins as given, and any great difference in the size of corresponding vessels in the two eyes, probably will prove to be abnormal.

Small red arteries sometimes appear at the temporal periphery of the papilla and pass into the retina toward the macula. These are the **cilioretinal** vessels, which come from Zinn's vascular plexus instead of from the central artery. Occasionally they have been known to furnish enough nutrition to the small area of retina that they supply to maintain a slight amount of circumscribed vision when the circulation through the central artery had been cut off. A cilioretinal vein is seldom if ever seen.

An alternate contraction and dilatation of a vein at the point where it turns to join the central vessel often is visible in the eye of a young person. This **venous pulse** seldom is to be seen elsewhere, in more than one vein, or in any except young people, but it can be excited in any normal eye by pressing gently upon the eyeball with the finger. When such a pulse cannot be excited in this manner, and retinal hemorrhages are present, we conclude that there is an occlusion of a central vessel. The spontaneous appearance of this venous pulse in the eye of an old person leads us to fear a commencing sclerosis. An **arterial pulse** always indicates an imbalance between the general blood pressure and the intraocular tension, so it is met with in glaucoma, and in general morbid conditions in which the blood pressure is abnormally high or low.

Pigmentation of the Papilla

A deposit of pigment sometimes is seen on the papilla, but unless it is accompanied by other pathological symptoms we probably shall be correct in deciding it to be of physiological origin. Once in a great while we may meet with a case in which the amount of pigment is very great, as in the one reported by *Beard* of a very black negro girl whose disk, which was of a deep scarlet pink, but appeared to be light through contrast with the dark brown of the rest of the fundus,

was so obscured by pigment that it resembled a full moon in partial eclipse.

THE BACKGROUND OF THE FUNDUS

When we pass from the papilla to the rest of the fundus we usually find the background of the picture to be of a nearly **uniform** red, having a delicately stippled appearance, and a deeper tone at the macula and about the papilla where the pigmentation is most dense. This is the commonest type of fundus. The uniformity of color is due to the fact that the pigment of the pigment epithelium is so abundant that we are unable to see any of the choroidal markings through the retina. The color varies in depth from a light to a brown red, according to the amount of pigment present. A description of all varieties of shades would be not only impossible, but useless, for we need to note only the uniformity, with the exceptions of a deeper tone at the places mentioned, and to perceive any other localized area that may have a different shade of color.

In the second type the pigment in the pigment epithelium is not sufficient to conceal the markings of the choroid, and we find more or less of the background composed of interlacing red bands separated by dark interspaces. The red bands are the choroidal vessels. They cannot be mistaken for the retinal vessels as they appear to be flat, are much broader, have no light streaks, divide irregularly, form many anastomoses, and have no uniform direction, or else converge toward the periphery. Quite exceptionally a case is seen in which they converge toward the posterior pole. They are situated deeply, as can be seen when one is crossed by a retinal vessel. The arteries cannot be distinguished from the veins, and both appear to be bright red from contrast with the choroidal pigment that fills the intervascular spaces and forms the other characteristic marking of the choroid. This is the **tessellated** fundus, sometimes called the **tiger choroid,** and is to be differentiated from a morbid condition in which the pigment epithelium has been destroyed by some pathological process so as to leave the choroidal markings visible, by its regularity of design, its sharpness of outline, its uniformity of surface and color, the absence of accumulations of pigment in the retina and of other pathological symptoms, and the good vision of the eye.

In the **albinotic** type neither the retina nor the choroid contains

much pigment, so the choroidal vessels appear to be dark from contrast with the yellowish white interspaces. The general color of the fundus in this type approaches pretty nearly to that of the papilla. *Beard* describes as a "poor choroid" what seems to be a variety of this type in which the vessels are narrower and more sparse than normal, and the choroid as a whole seems to be thin and meager. *Cowgill* reported a case in which the background of the fundus of each eye was as white as the sclera with no traces of either the pigment or the vessels of the choroid; in this case the choroid appeared to be absent and the normal retina to lie directly on the sclera. The albinotic fundus is met with almost wholly in very light blondes, or albinos, and perhaps we should look upon it as due to an imperfect development of the choroid, which is more faulty in *Beard's* "poor choroid," and reaches its extreme in the absence described by *Cowgill,* but in all of these the retina may not be affected and the sight be very good.

We seldom see a purely tessellated or albinotic fundus, for, as a rule, the pigment epithelium contains enough pigment to conceal the choroidal markings, at least at the macula and about the papilla. Much more often we see a fundus which has a background of uniform red in one part, and a tessellated or albinotic appearance in another, because of an uneven distribution of the pigment; but when we meet with a circumscribed area in which the choroidal vessels are visible we have to study it carefully to determine whether it is physiological or the result of disease. If the choroidal vessels appear to be healthy, if there are no lumps of pigment to be seen, and if we can find no other evidences of disease of the retina, we assume it to be a normal condition.

THE MACULA LUTEA

About one and a half papillary diameters (1½ p. d.) to the temporal side of the head of the optic nerve and at a little lower level, is a horizontally oval area which has a deeper hue than the rest of the fundus, and is darkest in its center. This is the macula lutea. In its center is a funnel shaped depression called the **fovea centralis,** upon which the rays of light are focussed by the dioptric apparatus of the eye, as it is the point of most distinct vision. We are not able to see the fovea in an adult, as a rule, but its situation usually is indicated in young persons by a bright reflex, which may be round,

crescentic, triangular, or of almost any shape, according to the way in which the light happens to fall on the walls of the fovea.

LIGHT REFLEXES

We are likely to see a number of other light reflexes from the retina of a young person, though they are not often seen in older people. Bright bands or spots appear along the blood vessels, or elsewhere in the fundus, and change their forms or positions with the movements of the head or mirror. They are to be seen most distinctly by the direct method when the pupil is not dilated and our own vision is focussed a little in front of the retina. Under such circumstances we may see a bright line about one papillary diameter to the nasal side of the papilla, which runs parallel to its margin and is called **Weiss's reflex ring.** This was thought by *Weiss* to indicate a detachment of the vitreous and to be pathognomonic of myopia, but he was mistaken. It is simply a reflex of the light from the retina and indicates nothing abnormal. In this respect it is altogether different from the gray curved lines that are sometimes to be seen in about the same situation, which do not change with the movements of the mirror, are shadows cast by the margins of a posterior staphyloma in high myopia, and are known as the **Weiss-Otto shadow rings.**

Another retinal reflex which is fairly constant in young people, in whom it can be seen best by the indirect method, is a bright ellipse that surrounds the central part of the macula. Its inner border is seen to be sharply cut, but its outer varies with the play of light, so that in the same case it is sometimes broad, sometimes narrow, and occasionally it may be seen to flash out over the adjacent surface of the retina. This is the **halo of the macula.** It varies in size, and at times is nearly circular.

ABNORMALITIES OF THE FUNDUS

Abnormalities manifest themselves through elevations and depressions of the surface, indistinctness that ranges from a slight haze to opacity, accentuated sharpness of detail in which parts normally hidden may become visible, alterations in the sizes, contours, courses, and color of the blood vessels, and changes of color. The papilla may be swollen or excavated, its details may be hidden, hazy, or too

distinct, perhaps enabling us in the latter case to see the meshes of the lamina cribrosa, its vessels may be too large or too small, irregular in outline, extremely tortuous, abnormally curved or nearly straight, and be darker or lighter than normal, while the background may be too red, too pale, or have a yellowish, bluish, or greenish tone. Elsewhere in the fundus we may find elevations or depressions; a haziness of a portion of the surface that may deepen into a yellowish white or grayish opacity; white, black, or red spots of variable shapes and sizes; large green or blue spots; areas in which the choroidal markings have been made visible; while the retinal vessels may be increased or diminished in number and size, or may present irregularities of contour, as well as too pronounced, broken, or lost light streaks. When the choroidal vessels are visible we may find a few or many to have been changed into yellowish white bands that seem to contain no blood, or in which the blood forms a narrow red line.

To make a diagnosis we take the changes present in any given case and consider their relations to one another, as well as to the parts of the fundus which remain normal, to the subjective symptoms, to the results obtained by testing the vision, the refraction and the visual fields, to the history, and to the general state of health or disease of the organism. We have to distinguish between congenital and acquired defects, as well as to recognize the presence of disease, locate the principal site of the latter as in the optic nerve, the retina, or the choroid, and to ascertain its ætiology. The entire picture of the fundus, so far as we can see it, must be taken into account, and we shall find at least two of these tissues affected in almost every case. Yet the signs which we accept as indicative of a lesion in one usually preponderate enough to lead us to conclude that this is the tissue chiefly affected.

CHAPTER XVI

The Papilla

The first thing we look for in the fundus is the papilla, because it forms a convenient center from which to make our other observations, and so it is the first part to receive our attention. We have to consider its color, its margins, its level, and its vessels, all in connection with the subjective symptoms presented. If it is much too large, or has a tag or cord of tissue projecting from it out into the vitreous, we recognize a congenital fault. If it seems too red or too pale, we must decide whether this is the effect of contrast with the color of the rest of the fundus, or of a pathological condition. If it is too pale, and this cannot be explained through contrast, we notice whether the appearance is produced by a large physiological excavation, and if not, whether the paleness is confined to the temporal sector. Well marked changes, such as a grayish, bluish, or greenish tint, or a hazy obscuration of the disk with enlarged, tortuous veins, are perceived at once, but slight deviations from the normal in the way of haziness of the margins, changes of level, and alterations in the vessels are apt to need close scrutiny for detection.

COLOBOMA OF THE OPTIC NERVE SHEATH

It is always possible to meet with an extreme rarity, so it may happen that a bluish gray papilla which is two, three, or four times as large as normal may appear unexpectedly before us. It will have a large excavation with precipitous walls, over the edges of which the vessels hook and disappear, just as in glaucoma. This excavation may be central, but more commonly it is found in the lower part of the papilla in contact with its margin, which is apt to be scarcely discernible when this defect is associated with an inferior conus, or with a coloboma of the choroid. The diagnosis is made readily from the enormous size of the disk.

A still greater rarity is the presence of a coloboma of this nature

in a papilla of normal size. We find a deep depression in the lower part of the disk, over the margin of which the lower retinal vessels hook, which can be differentiated from a glaucomatous cup only by the absence of any other signs of glaucoma, and the nonprogressive character of the condition.

PERSISTENT HYALOID ARTERY

When we see a cord extending from the center of the papilla to the posterior pole of the lens, or a tag of tissue that extends from the center of the disk into the vitreous, and perhaps oscillates slightly, though it causes no fluctuations in vision, we are accustomed to say that it marks the remains of a fetal hyaloid artery, although this origin is not certain in all cases. If it comes directly from the central artery, is filled with blood, and does not turn back on itself to form a twisted loop, there is no doubt as to the correctness of this diagnosis, and there is none if a cord extends from the papilla to the lens, but an opacity may persist after the absorption of an exudate into the vitreous which is hard to distinguish from a nonvascular remnant of the artery which floats out into the vitreous. *Oatman* says that such a case may be differentiated by the fluctuations that occur in vision, but I have been unable to convince myself that this is a positive indication. A twisted arterial loop that rises from and returns to the papilla should never be mistaken for a persistent hyaloid artery.

OPTIC ATROPHY

When the vision of an eye is poor or lost, the visual field contracted, the color sense impaired, the papilla distinctly white, yellowish, gray, bluish, or greenish, and no glaucomatous cup is present, the diagnosis of atrophy is easy, but these symptoms are not always well developed, and the atrophy may be either complete or partial. In a **partial** atrophy the fibers of a single bundle alone are involved, at least primarily. Practically the only example of this that we meet with is seen in disease of the papillomacular bundle, but a commencing total atrophy may be hard to distinguish from a partial by the ophthalmoscope alone, because the increasing pallor is most noticeable in the temporal segment of the disk, which is the palest portion normally, and is where the pallor of partial atrophy makes

itself manifest. The diagnosis of optic atrophy is not enough; this is simply a symptom induced by other trouble, and we should seek to ascertain its cause. This is to be learned mainly from the history and the other symptoms exhibited by the patient, but in total atrophy we gain some help at times from the picture presented by the disk itself.

Disease of the Papillomacular Bundle of Nerve Fibers

When a patient complains that he has been losing his vision gradually, and can get no satisfactory benefit from glasses, while the only objective lesion to be found in his eyes is a pallor of the temporal sectors of both papillæ, the margins, level, and vessels of which are normal, we have to deal with either a commencing total atrophy which is about equally advanced in the two eyes, or a disease of the papillomacular bundles of nerve fibers. We determine which of these two conditions is present through a study of the visual fields. If the peripheries of the fields are contracted and there is no central scotoma, the condition is one of commencing total atrophy, but if the peripheral limits are normal and a central scotoma can be demonstrated, even though only for red and green, the papillomacular bundles are diseased.

The **central scotoma** at first is for red and green alone, but as the disease progresses white fails to be perceived and the scotoma becomes larger. The prognosis depends to a certain degree on the scotoma, for as long as the central vision is impaired but not lost, recovery is possible, but after any of the fibers have actually become atrophic it is doubtful if they can ever regain their functions. When the disease has been arrested after a certain amount of atrophy has taken place, we may find many years later enduring marks in the form of a sharply defined white temporal sector of the papilla, and a central scotoma. If it is unchecked the disease is likely to spread to the other fibers of the nerve and to develop a total atrophy, but the peripheral vision will be the last to go. Occasionally a central scotoma with a contraction of the periphery of the field seems to show that while the papillomacular bundle is affected the most, the other fibers of the nerve are involved.

All hope of successful treatment depends on the discovery of the **cause.** We inquire rigidly into the habits of the patient, for the commonest cause is chronic poisoning with alcohol and tobacco. We

also inquire into his occupation, for less often it is produced by poisoning with iodoform, cannabis indica, lead, or bisulphide of carbon. In the last case the field is apt to be contracted. Although toxic causes predominate we should never fail to investigate the history and the central nervous system for symptoms of multiple sclerosis, in which this affection is about as common as total atrophy, or optic neuritis, even though it is not present as a rule. The posterior accessory sinuses also must be investigated, as cases of this nature have been reported as caused by inflammation in the posterior ethmoidal cells and in the sphenoidal sinus.

Neuritic Optic Atrophy

When the papilla is very white or gray, perhaps with a bluish or greenish tint, and seems smoothly opaque, its margins blurred, or slightly irregular if distinct, the arteries small, some of them possibly with sclerotic walls, the veins tortuous and either disproportionately larger than the arteries, or small, we not only recognize an optic atrophy, but we infer that the atrophy followed an optic neuritis or a choked disk, and therefore call it neuritic. Changes in the retina and the choroid frequently are to be found elsewhere in the fundus. The ætiology is that of the optic neuritis, or the choked disk, of which this forms the final stage.

Simple Optic Atrophy

When a patient gives a history of sudden blindness, or of a gradually increasing impairment of his color sense and then of his vision for both distance and near, together with a gradual contraction of his visual fields, that perhaps has ended in total blindness, and we find a pale papilla, which may vary in color from a pearly bluish or greenish hue to chalky white, probably dotted with little gray spots that mark the meshes of the lamina cribrosa, with a margin that is very sharply defined, as a rule, but occasionally may be a little blurred, a level that is normal or presents an atrophic excavation, and vessels that may be normal or small, but are never engorged or unduly tortuous, we know that the atrophy did not follow an engorgement of the papilla, and so call it simple. Some writers confine the term simple to those cases in which tabes is present and call all others secondary, but the two can hardly be distinguished ophthalmoscopically.

Such an ophthalmoscopic picture does not necessarily prove the presence of atrophy, for a healthy papilla may be very pale in anæmic or old persons in whom the vision is good, so the vision must always be tested before the diagnosis is made.

A simple optic atrophy that appears soon after a sudden blindness may be due to laceration or compression of the nerve fibers through traumatism, or to poisoning. One that develops slowly must be due to a gradually increasing pressure in the front part of the skull, or to a slowly progressive lesion of the nerve itself, which may have been caused by traumatism, by poison, or by disease.

Sudden Pallor of the Papilla

A shot or stab wound of the orbit that causes immediate blindness followed in a few days by optic atrophy, has severed the nerve. Blindness immediately after a traumatism to the head, followed in a short time by simple optic atrophy, indicates that the base of the skull has been fractured and that the nerve has been lacerated or compressed by a fragment of bone. When an attack of blindness, or of impaired vision, is sudden, is associated with dilated, rather irresponsive pupils, and the pallor of the disk appears more quickly than is usual after traumatism, of which there is no history, we suspect poisoning with quinine, or with wood alcohol, conditions which are described under toxic amblyopia.

Gradually Developing Pallor of the Papilla

Oxycephalus may furnish a sufficient explanation of a gradually increasing pallor of the disk in a child, for the optic nerves are apt to suffer as the bones grow in this deformity of the skull.

During the course of our examination our attention may have been attracted by a myosis, a reflex immobility of the pupils, perhaps an anisocoria, or a paresis of some of the extrinsic muscles, so that when we find a simple optic atrophy we test the patellar reflex in full expectation that we shall find it absent, and feel sure of the diagnosis of **tabes dorsalis.** This disease is the commonest cause of simple optic atrophy, as it is responsible for nearly one half of all of the cases we meet with. The degeneration is supposed to begin in the ganglion cells of the retina and to proceed up the nerve. The first symptom is a contraction of the fields for color, particularly for green; then the field for white grows smaller and the central

vision fails until blindness supervenes. The lapse of time between the first symptom and the extinction of vision varies, but the average is said to be two or three years. Often the time is shorter, sometimes it is longer, and rare exceptions are on record in which some vision was retained for many years, until the death of the patients. The nature of the contraction of the visual field is not always the same, sometimes it is concentric, sometimes it takes the form of sectors, but invariably the periphery is affected the most, and we do not commonly find a scotoma in tabes. No true hemianopsia is produced, though occasionally we meet with a case in which one is simulated by a uniform contraction of both fields on the temporal side. A true hemianopsia in a case of tabes indicates the presence of some intracranial complication. Sometimes both eyes are affected simultaneously, but more frequently one suffers before the other, and the blindness of the first eye may not be noticed until the sight of the second begins to fail.

Although tabes is suggested very strongly by a reflex immobility of the pupils with a simple optic atrophy, we must be on our guard, for it is possible that we shall elicit symptoms that lead instead to a diagnosis of **general paresis,** or less often to one of **cerebral syphilis,** or of **multiple sclerosis.** When the pupils are dilated, perhaps unequal in size, and are not responsive to either light or convergence, a simple optic atrophy seldom is due to tabes. If the patient has palsies of the muscles of his face or limbs, delusions of grandeur, or attacks of mania, the disease is more likely to be general paresis. If he has internal ophthalmoplegia, whether it is combined or not with an external one, has a bilateral ptosis, or has hemianopsia, and particularly if the optic atrophy is confined to one eye, we shall probably find the case to be one of cerebral syphilis. If he has nystagmic twitchings of his eyes, pareses of certain of their muscles, and especially a paresis of their associated movements, we are guided toward multiple sclerosis.

Sometimes we find a simple optic atrophy with no other abnormal condition of the eyes, for all of the ocular symptoms, including this one, may be absent in any of the diseases that have been mentioned. In such a case we need to investigate the organism not only for symptoms that may be referable to these diseases, but also for any that point toward any **intracranial condition** which may cause pressure on the optic nerve, such as a slowly growing tumor at the base of the brain, a hydrops of the third ventricle, or an arterio-

sclerotic carotid. Symptoms of acromegaly sometimes locate the cause as a tumor of the hypophysis. Rarely a simple optic atrophy accompanies congenital spastic paralysis, Little's disease of the spinal cord. Occasionally we are unable to discover anything that is indicative of injury or disease, and then we are obliged to wait for the development of other symptoms. We know that a simple optic atrophy may be the first, and for a long time the only symptom of tabes to appear, and a positive Wassermann will greatly strengthen our suspicions in this direction, but no positive diagnosis can be made from these indications alone.

A simple optic atrophy in which the **vessels are threadlike** leads us to look for other indications that the central artery has been occluded in the past if only one eye is affected, or that the patient has been poisoned with quinine, or wood alcohol, if the condition is bilateral. The pictures produced by permanent atrophy caused by quinine, and by bilateral occlusion of the central artery, can be differentiated only by the history, but bilateral occlusion of the central artery is rare.

When the color of the papilla is yellowish rather than white, the margins are indistinct, and the vessels are threadlike, we shall be almost sure to find elsewhere in the fundus lesions that are characteristic of pigmentary degeneration of the retina, or of an old choroiditis, so this form of simple atrophy is called **retinitic.** This peculiar condition of the papilla, taken together with a concentrically contracted field and hemeralopia, suffices to establish the diagnosis of pigmentary degeneration of the retina in atypical cases in which the characteristic black spots are absent.

Hereditary Optic Atrophy

When we see a case of optic atrophy which has no apparent cause in a young man, and inquire into his family history, it is quite possible that we shall learn that male members of his family have suffered in the same way for generations. The females seldom suffer themselves, but they are apt to transmit the taint. *Norris* has given us the histories of two such families, traced through five generations in one, through four in the other. He tells us that the first subjective symptom is a central scotoma for color that increases until light perception is lost in the affected area. Frontal headaches and a cloud before the eyes frequently are prominent symptoms. The

field is contracted, but complete blindness is rare. He describes three stages: first that of a cloudy swelling and œdema of the papilla, second that of discoloration, third that of atrophy. *Oatman* says that the atrophy is simple, *Norris* that it is neuritic. The onset may appear at any time from childhood to middle life, but occurs most commonly after puberty and in early manhood. Cases of partial recovery have been reported.

REDNESS OF THE PAPILLA

When the papilla seems to be redder than usual the chances are that it appears so because of contrast, of the nature of the light employed, or of a hyperæmia that is of no great importance, provided that its margins, level and vessels are normal. A **hyperæmia** of the papilla with dilated retinal vessels is met with in general plethora, in fevers, and after overindulgence in liquor, but may be caused also by accommodative efforts in hypermetropia and presbyopia, by any traumatism to the eyeball, by an inflammation anywhere in the eye, by glare and heat, by an examination with the ophthalmoscope, by obstruction to the general circulation, as in some forms of heart disease and when the great vessels are compressed, by certain disorders of the brain, and is seen in some types of insanity. In addition it may be a symptom of an inflammation of the middle ear, or of the accessory sinuses of the nose, but, as a rule, it is only when other symptoms lead us to look for an optic neuritis, a choked disk, or a glaucoma, that a hyperæmia of the papilla is important as the first ophthalmoscopic sign of such a condition. If the patient has a purulent otitis media a hyperæmic disk surrounded by an œdematous retina leads us to fear an intracranial complication, probably sinus thrombosis.

When a papilla looks redder than it should and has margins that are more or less obscured, we must note its shape and level, the condition of the retinal vessels, the clearness or obscuration of the periphery of the fundus, test the vision and the fields, and sometimes measure the refraction, to ascertain whether the appearance is due to an affection of the optic nerve or not, and if so whether the nerve head is inflamed or engorged with lymph.

A beginner may be troubled because the entire disk appears to be hazy, and be inclined to suspect an optic neuritis when the truth is that the papilla is not quite in focus. He must ascertain whether

this is the case by moving his head back and forth slowly, if he is using the indirect method, or by interposing lenses in the ophthalmoscope, if he is looking at the upright image, until he has obtained the clearest picture possible. If this persists in remaining hazy he must learn whether the haziness extends to the periphery of the fundus or not before he can say there is trouble in the nerve head, for when we see a red papilla that shines as it were through a cloud which spreads over the adjacent retina so as to render all of the details indistinct, and cannot be dissipated by focussing, we have to determine whether the picture is caused by a diffuse opacity of the refractive media, by a pseudoneuritis, or by an optic neuritis. The vision is impaired in all of these conditions.

If the periphery of the fundus is just as hazy as the center, we are trying to see through a diffuse opacity of the media, for the obscuration associated with an optic neuritis, or a choked disk, extends outward only a short distance from the papilla. Inspection will inform us whether the opacity is in the cornea or the lens, and if it is in neither of these it must be in the vitreous. Still it is quite possible for an optic neuritis to coexist with a diffuse opacity of the media, so we have not finished by finding the latter. We look to see whether the retinal veins are unusually broad and tortuous, and whether any hemorrhages or white spots can be seen in the retina, for if we can find any of these things we know that an optic neuritis is present. Otherwise such a diagnosis is rendered probable only by the detection of a central scotoma.

If the details in the periphery of the fundus are clear we conclude that there is no diffuse opacity of the media, and next consider the possibility of a **pseudoneuritis.** Should the papilla appear to be remarkably oblong in shape, while the obscuration of its margins does not seem to extend out into the retina, where we can find no hemorrhages or white spots, and should the visual field be normal, we suspect that the appearance of the disk is caused by a high degree of hypermetropic astigmatism, usually compound, even though the vessels may look as though they were engorged and tortuous, and the surface of the papilla seem to be elevated a little. This suspicion becomes certainty if we find that by slowly adding stronger and stronger plus lenses in the ophthalmoscope two opposite margins of the disk are caused to become distinct, together with the vessels that run in the same direction, and that by continuing to change the lenses these edges and vessels are caused to fade away while others

come into view until those which are at right angles to the ones first seen are distinct. The refractive error can be measured in this way if the observer's own accommodation is under perfect control.

After these conditions have been excluded we have to determine whether we have to deal with an optic neuritis or a choked disk. Many excellent writers and observers confound these two conditions, and make no distinction between an inflammation in which the papilla is exceptionally swollen and an engorgement of the papilla with lymph. This seems to be because it is customary to speak of the height of the swollen papilla as the distinctive characteristic of a choked disk, although the effect produced on the ***vision*** seems to be of more value in the differentiation. A choked disk may be low in its early stage, or during involution, and its maximum height is no greater than that which is observed occasionally in optic neuritis, but in the former the vision is apt to remain good for a long time in spite of the formidable appearance of the lesion. After viewing an enormously swollen papilla, with its engorged and tortuous blood vessels, we may be amazed to find the vision normal, but more commonly we find it somewhat reduced, with temporary attacks of blindness or of obscuration, and with various faults in the field, but rarely if ever with a central scotoma, while in nearly if not quite every case of optic neuritis the vision becomes bad early in its course, and frequently a central scotoma can be demonstrated.

OPTIC NEURITIS

When a patient complains of a recent great impairment of vision, and we see a papilla that is redder at its center than at its margins, while the latter are obscured by a gray or reddish cloud that shades off into the surrounding retina, or see a disk that looks like a red sun seen through a fog, or one that is so blotted out that we can locate it only by the convergence of its blood vessels, we do not hesitate to pronounce the case one of optic neuritis if the veins are broad and tortuous, the arteries are normal, and hemorrhages or white spots are visible elsewhere in the fundus. The surface of the papilla may be on a level with the retina, in which case the physiological excavation almost certainly is filled up, but, as a rule, it is elevated. The hemorrhages and white spots may be found on or about the papilla as well as elsewhere, and are particularly apt to occur about the macula. When the papilla is greatly swollen the

veins are very broad, distended and tortuous, dipping in and out of the cloudy tissue, hidden in some places, appearing in others as disconnected pieces, while the arteries may be so buried as to appear smaller than normal, and the gray cloudiness may have radiating striations, so that the picture is exactly the same as that presented by a typical choked disk. The impaired vision, the central scotoma if one can be detected, and the discovery of the ætiology settle the diagnosis.

An optic neuritis may be simulated more or less closely by inaccurate focussing, by a pseudoneuritis, by a supertraction crescent, by an occlusion of the central artery, by a flat detachment of the retina, and by a traumatic œdema of the retina. The first two have been dealt with. A supertraction crescent is differentiated by the normal vessels and the presence of a myopic conus or staphyloma; an occlusion of the central artery by the sudden blindness, the broad gray expanse in the retina, and the cherry red spot in the fovea; a flat detachment by the local elevation of the retina, the absence of the light streaks on the vessels that pass over it, the discoloration of this area, and a corresponding defect in the visual field; a traumatic œdema by the history of traumatism, the normal vessels, and the rapid recovery.

CHOKED DISK

When in spite of good vision we find a reddish gray papilla that is enormously swollen and elevated and has precipitous sides, with arteries that are smaller than normal, and veins that are very broad, dark, and tortuous, has a great many small vessels on its surface, and has its base surrounded by an opacity with radiating lines that covers its margins and extends out so as to make the papilla appear to be abnormally large, we diagnose a choked disk.

The **height** of the swelling usually is 1 mm., 3 diopters, or more, though it may be much less. Its surface, as well as that of the opaque retina in its immediate vicinity, has a striate, reddish white appearance that corresponds to the courses of the bundles of nerve fibers. The retinal arteries are quite small, while the broad veins bend about in great loops. Often the great vessels can scarcely be seen in the center of the papilla, but come into view in its sides, or at its margins. The veins may be accompanied by white stripes along their sides. In children and young people we frequently see

small, brilliant white spots, or fine brilliant white lines, on or outside of the papilla, while hemorrhages and white spots are present almost invariably in both the disk and the retina.

In the early stage of a choked disk the entire papilla may be swollen to a less degree, or only a part of it may be swollen. Occasionally the periphery may be seen to protrude above the center. Its size may seem to be increased by the œdema that obscures its margins and infiltrates the adjacent retina. It may have a clear appearance, the vessels may be only slightly changed, and the retina may appear to be normal, except for a narrow rim about the papilla, even when the top of the swollen disk is two thirds of a millimeter above its proper level. The differentiation from an optic neuritis in such a case and at this time, is made much more easily through the disproportion of the swelling to the retinal changes, the disproportionate sizes of the arteries and veins, and the presence of good vision.

Finally the vision fails, the redness of the papilla is replaced by pallor, the swelling subsides, and a neuritic atrophy supervenes. The subsidence may affect only one part of the papilla at first, just as one part may be the first to swell. Exceptionally a choked disk of short duration subsides without inducing atrophy.

Diagnostic Value of an Optic Neuritis

Optic neuritis is not a disease, it is only a symptom, and our real diagnosis has not been made until we have discovered its cause. It may be due to a malformation of the skull, to an inflammation in the orbit, or in the accessory sinuses, to disease of some of the internal organs of the body, including the vascular and central nervous systems, to an acute infectious disease, to a disturbance created by irregular or suppressed menstruation, lactation, or loss of blood, to poisoning, or to sympathetic inflammation. The only cases in which an optic neuritis may be said to be a pathological entity are the very rare ones in which it attacks a large proportion of the male members of a family about the age of puberty for several generations, and results in hereditary optic atrophy, but our knowledge of these is slight.

When an optic neuritis occurs during the course of one of the **acute infectious diseases,** such as measles, scarlet fever, diphtheria, whooping cough, typhoid fever, influenza, pneumonia, facial ery-

sipelas, gonorrhea, malaria, polyneuritis, or acute articular rheumatism, we are justified in ascribing it to the disease itself, but later it may be due to a sequel, like nephritis, or an inflammation of the accessory sinuses, or to a different infection, like syphilis. It is said to have followed catching cold, and exposure to severe weather, but such a cause as this cannot be accepted until every other one has been excluded.

We can never infer the cause with any certainty from the ophthalmoscopic picture, although in some cases it is quite suggestive; no matter what suspicions the appearance may engender, they must be confirmed or disproved by a searching investigation of the organism. Occasionally we may be guided toward a diagnosis of **syphilis** when a wide area of the retina about the papilla is occupied by a dense œdema with radiating hemorrhages, with or without white spots, there is a diffuse central opacity of the vitreous, or patches of disseminated choroiditis are to be seen in the periphery of the fundus, but these signs are wanting in many cases of luetic neuritis.

When the patient is a child a glance at the conformation of the skull may inform us of a probable **malformation of the orbit,** and it is well to inquire into the family history in search of a hereditary predisposition, but the chances are that an optic neuritis in a child is tuberculous. We must search for other clinical symptoms of tuberculosis, especially in the lymphatic glands, and observe the effect produced by the tuberculin test. Sometimes when the patient is very sick we may see small, roundish, elevated, yellowish white spots in the fundus, or an elevated white mass in the papilla, which we take to be tubercles and render the diagnosis almost certain.

When the patient presents symptoms indicative of meningitis, abscess of the brain, or sinus thrombosis, the optic neuritis is explained, but we still have to learn the cause of the intracranial trouble. If there is considerable œdema of the retina about the papilla with vessels that are very slightly changed, our attention is directed to the middle ear. Exceptionally a disseminated choroiditis, or tubercles in the choroid, suggest syphilis, or tuberculosis.

If the retina contains many small hemorrhages and white spots, especially about the macula, we suspect nephritis, or ascribe it to diabetes if the patient is in a late stage of this disease. No one picture can be said to be characteristically produced by either of these diseases. The retinitis usually predominates over the neuritis, but the opposite may be the case and the swelling of the papilla may be

so great as to simulate in almost every respect a choked disk, and as this is met with most frequently in cases of nephritis the condition is commonly known as **albuminuric choked disk.** At the same time the many small hemorrhages and white spots, even in their so-called characteristic grouping, have been observed in neuroretinitis from other causes. One symptom, which is quite suggestive of diabetes rather than of nephritis, when it is detected in these cases, is a very small central scotoma.

An optic neuritis in an old person may be due to **arteriosclerosis,** but this form presents no characteristic signs, unless we have the opportunity to watch its development, which is very slow. Occasionally the swelling of the papilla is enormous when it resembles very closely an albuminuric choked disk.

An optic neuritis in only one eye generally is due to an **inflammation in the orbit.** A wound, a cellulitis, or a periostitis of the walls near the apex, gives rise to symptoms that attract attention prior to the examination of the fundus, but this is quite apt not to be the case when it is caused by **inflammation of the posterior ethmoidal cells,** or **of the sphenoidal sinus.** These accessory sinuses should be investigated in every case in which the cause of an optic neuritis, a disease of the papillomacular bundle, or a choked disk, is doubtful, whether it is present in one or in both eyes, but especially when it is confined to one, because inflammation there may ruin the eyes before it excites any other subjective symptom. We should also take note of any persistent dropping of a watery fluid from the nose.

When an eye has been wounded at some previous time and is tender to the touch, an optic neuritis that develops in the other may be of **sympathetic** origin. This diagnosis is confirmed if we can find little yellowish spots in the periphery of the fundus, and some signs of a uveitis, such as diffuse opacities in the vitreous, deposits on Descemet's membrane, or evidences of iritis.

Diagnostic Value of Choked Disk

A choked disk of **only one eye** ordinarily indicates a lesion that compromises its optic nerve, while one of both eyes is an evidence of increased intracranial pressure. In the majority of cases of unilateral choked disk signs of trouble in the orbit have attracted attention before the discovery of the condition of the papilla, and

furnish a sufficient explanation. An abscess or a tumor in the frontal portion of the brain may break through into the orbit and produce the same local symptoms, which are not always accompanied by those of brain trouble. If the orbit is normal we must remember that a single choked disk is sometimes, though quite rarely, caused by a tumor at the base of the skull, by a hemorrhagic pachymeningitis, by an otitic sinus thrombosis, or by a hemorrhage at the base, but we should never fail to make a thorough exploration of the sphenoidal sinus, and of the posterior ethmoidal cells, for it is here that we are quite apt to find the cause.

A **double** choked disk is a symptom produced by a tumor of the brain in most cases, though not in all. The next most common cause is the distention of the cerebral ventricles in hydrocephalus, but there are others that are much less common, among which are a congenital malformation of the skull, abscess of the brain, sinus thrombosis, chronic meningitis of the base, and hemorrhages into the subdural or subarachnoidal spaces at the base of the brain. We must also bear in mind that the picture of optic neuritis presented in some cases of nephritis, arteriosclerosis, and of various acute infectious diseases, cannot be distinguished ophthalmoscopically from that of choked disk.

Choked Disk with Tumors of the Brain

When we find a double choked disk in a patient who is suffering from headache, dizziness, and vomiting, who has a slow pulse, mental or cerebral disturbances, and other general and ocular symptoms, we feel sure that he has a tumor of the brain. There is nothing in the ophthalmoscopic picture that can give us a clue to the nature or the situation of the growth, which may be a sarcoma, a glioma, a tubercle, a gumma, a cyst, or an aneurysm, and may be situated in the substance of the brain, in the membranes, or in the bones. Sometimes symptoms of tertiary syphilis lead us to believe the growth to be a gumma, or those of tuberculosis suggest that it may be a tubercle, and once in a while these suspicions are confirmed by the results of treatment, but in the great majority of cases the nature of an intracranial neoplasm must remain a matter of conjecture until after it has been removed. Its situation must be learned, if at all, from the general symptoms, which include among others convulsions, local spasms, palsies of various muscles, including the ocular and the associated movements of the eyes, contractures, ataxia, dys-

phagia, polyphagia, neuralgias, areas of anæsthesia, hyperæsthesia, or paræsthesia, disorders of hearing, taste, and smell, trophic troubles, diabetes mellitus or insipidus, and albuminuria. In any given case these symptoms may be few or many, slight or severe, brief or protracted, constant or paroxysmal, but it is from the combinations which they form that we are able to locate the lesion in some cases. It is only in a few of these that the ocular symptoms furnish the differential characteristics.

Although choked disk occurs in ninety per cent. of all cases of tumor of the brain, its absence from the syndrome during the early stage is in no way disconcerting, as it is apt to be a **late symptom.** When it does not appear at all, and no localizing symptoms are presented, we incline to locate the tumor in the anterior part of the brain, because the general symptoms induced by a tumor in this locality are vague, and because the appearance of a choked disk seems to be the more certain the farther back the tumor lies. No conclusions as to the size of the tumor can be drawn from the height of the swelling of the disk, as a very small growth may give rise to an enormous engorgement, while a large one may cause little or none. Temporary attacks of blindness, or of obscuration, likewise are of little diagnostic help.

Once in a while we see at an early stage in the course of a tumor a double choked disk that is **atypical** in that it is associated with a serious impairment of vision and a retinitis, so that it resembles exactly an albuminuric choked disk, but is attended by enough of the above mentioned symptoms to make its ætiology clear. If the patient has an anæsthesia of the cornea, nystagmic twitchings of the eyes, divergent strabismus, an inability to turn the eyes past the middle line toward one side, and a cerebellar ataxia, or a vertigo in which he feels as if objects were revolving about him, or he himself were revolving in space, we locate the tumor in the cerebellum. The anæsthesia of the cornea may be the first of all the symptoms to appear. If in addition to the above symptoms one external rectus is paralyzed, the tumor probably is on that side of the cerebellum and is pressing upon the abducens. Instead of these symptoms the patient may be found to have a central deafness of one ear, and a facial palsy on the same side, with a loss of equilibrium and ataxia; then our attention is directed to the angle between the cerebellum and the pons. When no such localizing group of symptoms accompanies an early choked disk of this nature we know that it can be

induced by a tumor at the base of the skull near the chiasm and the cavernous sinus.

When the tumor is situated elsewhere in the brain choked disk appears late as a rule, is associated with more or less good vision, and simply corroborates a diagnosis already made. In a few cases other ocular symptoms are of service in the **localization** of the growth, but often they are indefinite or absent.

If the patient has a paresis of some, or of all of the muscles of one eye that are supplied by the oculomotor nerve, associated with hemiplegia of the other side of the body, we look for a tumor in the cerebral peduncle. If the internal muscles of the eye are not affected the lesion probably is in the spinal portion, but if the ophthalmoplegia is total it is likely to be in the median part, or if sensory disturbances are added we suppose it to have extended to the tegmentum, while if the hemiplegia is replaced by ataxia we expect to find it in the lemniscus. If the muscles supplied by the oculomotor nerves of both eyes are paretic, or especially if the associated movements of the eyes upward and downward are paralyzed, while the patient has a central deafness of one ear and difficulty in standing or walking, the tumor probably is in the corpora quadrigemina.

Hemiplegia of one side and a paresis of the external rectus, or of the facial muscles, on the other, locates the tumor as probably in the pons on the same side with the paretic cranial nerve. If this nerve is the abducens we suppose the lesion to be at a place in the pyramidal tract through which the roots of the nerve pass. If the eyes cannot turn past the middle line toward one side, it probably involves the nucleus of the abducens on that side. This paralysis of the associated lateral movement of the eyes may be the only symptom present, in addition to the choked disk, or it may be combined with a loss of the power of convergence on the part of the affected internus, or it may be associated with any or all of the other symptoms produced by tumor of the brain. Paralysis of both sides of the body with the eyes fixed in the median line, unable to turn to either side, leads us to think that both sides of the pons have been invaded by the tumor.

Further than this we can get little help from the symptoms presented by the eye in locating a tumor of the brain, though occasionally the knowledge that isolated paralyses of muscles which are supplied by the oculomotor nerve may be caused by a lesion in one of the cerebral ganglia is of some service.

Choked Disk in Other Forms of Intracranial Trouble

The diagnosis of **hydrocephalus internus** is not difficult when the head of a child has grown disproportionately since birth, and this is a full explanation of the presence of a double choked disk. We seldom if ever meet with choked disk in hydrocephalus externus, and do not very commonly in any of the following troubles.

When a patient who is suffering from an **otitis media** develops a choked disk, or an optic neuritis, which occur about equally often, we apprehend an extension of the inflammation into the cavity of the skull, and the development of a sinus thrombosis, a meningitis, an abscess of the brain, or a subdural abscess. A choked disk is the more likely to be overlooked because the vision remains good, so perhaps it occurs more often than an optic neuritis, to which attention is apt to be called by the patient because of an impairment of vision. It is well to examine the fundus in all cases of purulent otitis in which symptoms lead us to suspect an extension of the aural disease to the brain. In rare cases a unilateral choked disk is found in the eye on the same side with the thrombosis. The special importance of these symptoms on the part of the papilla in these cases is that they indicate the need of an immediate operative intervention. After operation the condition of the papilla may undergo a rapid involution, but it is quite apt to grow worse for a time. A choked disk of this nature seldom results in atrophy.

A double choked disk that is more pronounced on one side than on the other, and in which the disproportion in size between the arteries and the veins is less than usual, is suggestive of a **hemorrhage** into the subdural or subarachnoidal space on the side of the more pronounced condition. Double exophthalmos calls attention to the cavernous sinus, obstruction of which by a thrombus or otherwise may produce choked disk. When the patient has been suffering from a very **debilitating disease,** the onset of choked disk may indicate a thrombosis of the longitudinal sinus; this is apt to be associated with spasmodic movements of the eyes, but not with exophthalmos.

CHAPTER XVII

The Retina and Choroid

When we come to study the rest of the fundus we find that we have to deal with conditions of pallor, redness, and haze that are not always well defined, with discolored areas, abnormal markings, white, black, and red spots of various shapes and sizes, elevations and depressions of the surface, and abnormalities in the blood vessels; and that from these we are expected to locate the lesion in the retina or in the choroid, and to ascertain its nature. With the exception of the rare cases in which a broad expanse of white sclera is visible through a functionating retina and we suppose the choroid to be absent, no lesion in the choroid can be visible as long as it is covered by a healthy retina, the pigment epithelium of which contains its usual amount of pigment. A lesion in the choroid makes itself manifest through a change in the overlying retina; this change bears a close resemblance to one produced by trouble in the retina itself, and there is no rule that is applicable to all cases by which we can differentiate between retinal and choroidal troubles. Consequently it is not an easy matter to deduce their nature and origin; on the contrary the attempt to do this affords an opportunity for wide divergence of opinion, even on the part of the most skilled and experienced, and in many cases our diagnoses might with more propriety be called simple inferences. The best that we can do is to observe all of the changes closely, study their relations to one another and to all other visible parts of the fundus, to consider them in connection with the history of the case, the subjective symptoms, the results of our tests of the vision and of the fields, and the known pathological findings in similar cases, and then to deduce the origin and nature of the lesion which produced them. When we have done this to the best of our ability we are justified in dignifying our inference by calling it a diagnosis.

ANÆMIA OF THE FUNDUS

When the whole fundus seems to be paler than usual, and the vessels to be rather small, we can only suspect anæmia, unless the

pallor is so great as to approach ischæmia, because the shade of color of the fundus differs so much normally in different persons. If we can detect the least haziness we appreciate that the pallor is one of the signs of a retinal œdema. If there is no haziness we consider whether the patient presents any other symptoms of general or cerebral anæmia, syncope, chlorosis, pernicious anæmia, or leucocythæmia. Retinitis is apt to be present in the last, though it rarely is met with in the other conditions.

By **ischæmia** is meant a sudden intense pallor of the fundus associated with an extreme smallness of the blood vessels and a sudden partial or total blindness, that may be temporary. It is observed most often just after an occlusion of the central artery, and in cases of poisoning with quinine, ergot, or salicylic acid, but is seen occasionally in epileptiform convulsions, whooping cough, malaria, cholera, erysipelas, and sick headache.

HYPERÆMIA OF THE FUNDUS

Hyperæmia of the fundus is as difficult to recognize as anæmia, for the same reasons. If we are acquainted with the previous appearance, or if the change is confined to one eye, the difficulty is not so great. We may perceive that the vessels have become larger and more tortuous, and that the fundus is reddened by a capillary congestion, when a hyperæmia appears during an ophthalmoscopic examination, but ordinarily we notice simply a reddened papilla with a retinal striation about its slightly blurred margin. This appearance must be taken in connection with the other conditions of the eye, the physical condition of the patient, and his habits and vocation, before we shall be justified in pronouncing it hyperæmic.

Active hyperæmia often is a symptom of retinitis, but when no signs of this inflammation are to be found it may have been caused by eyestrain due to refractive errors, to muscular imbalance, or to overwork in a bad light, or it may have been brought about by exposure to strong light or heat, by inflammation elsewhere in the eye, or by an increased supply of blood in fever. **Passive hyperæmia** may be caused by any obstruction to the outflow of the venous blood, whether it affects the general or the local circulation, and may be so great as to amount to cyanosis; such a congestion accompanies glaucoma and choked disk, as well as many bodily ailments in which the venous circulation is impeded.

SPOTS IN THE FUNDUS

A **large white** or grayish area may call our attention to a patch of medullated nerve fibers, to an ischæmia, œdema, or necrosis of a part of the retina, to a sclerosis of the choroid, to a conus, to a posterior staphyloma, to a coloboma of the choroid or of the macula, or to a lesion produced by choroiditis. **Small white** spots may or may not be of pathological significance. A **large greenish** or **bluish** area suggests a detachment of the retina, so we look immediately at the blood vessels to learn from their courses whether there is a change of level. Depressions in the fundus are met with in only a few conditions, but elevations are common, and may be slight or enormous.

Black spots are formed by the aggregation of particles of pigment that have been set free from the cells in which they are normally contained. They are common about the papilla, and occasionally one or two may be seen elsewhere in the fundus of a normal eye, but in such cases they are not attended by any signs of disturbance. In morbid conditions they indicate that the pigment epithelium of the retina has been destroyed, and we have to determine as well as we can from the accompanying symptoms whether the lesion is situated mainly in the retina, or in the choroid.

In very rare cases a **red** spot may happen to be caused by a congeries of minute blood vessels, but in the great majority it marks a hemorrhage. In some cases the spot is so small and light as to escape notice unless the inspection is made very carefully, while in others it is so large and dark as to resemble a pool of blood. Its color depends chiefly on its age and thickness, and gives no information as to whether the blood came from an artery or a vein. When the hemorrhage is fresh the usual color is a bright blood red, but later it assumes a dark brownish tone, and then it grows darker until it disappears, or is changed into a white spot that finally passes away. Pigmentation seldom remains to mark the place where the hemorrhage took place, and the blood rarely gives rise to a formation of connective tissue.

Choroidal Hemorrhages

Almost every hemorrhage that we see is in the retina, but in very rare cases, when a hemorrhage occurs beneath the choroidal vessels in an albinotic fundus, we can recognize with certainty that

it is in or beneath the choroid. In the case described by *Loring* the hemorrhage was linear and extended from the anterior part of the field along the inner wall of the eye nearly to the papilla. It is rather customary to describe extravasations into the choroid as forming red or brown, oval or circular spots well beneath the retinal vessels, but it must be admitted that such a spot is quite as likely to be situated in the deep layers of the retina. *Lawson* says that a hemorrhage situated between the choroid and retina elevates the latter so that the retinal vessels can be seen to curve over it, and *Loring* suggests that when such a hemorrhage happens to occur near the posterior pole of the eye its location may be at least inferred from the presence or absence of a scotoma, as this will be absent only when the retina is not affected. Most choroidal hemorrhages that have been observed were of traumatic origin, but it is said that some have been known to occur in myopia, and in eclampsia.

Retinal Hemorrhages

The **shape** of a hemorrhage into the retina is irregular and may be said to vary infinitely, but it presents characteristics from which we are able in many cases to form an opinion as to its **depth.** If it is irregularly round and looks lumpy, we believe it to be in the deep layers; if it is drawn out along radii from the papilla, no matter whether it is ribbon shaped, fusiform, or has points that appear as though it had been teased out longitudinally, we place it in the layer of nerve fibers; if it covers one or more vessels, it is in the superficial part of this layer; if a vessel associated with it is swollen so as to have a spindle shape, we suppose that a part of the hemorrhage has distended its sheath; while a round or oval red disk, the upper margin of which may be a straight horizontal line, can be pronounced confidently to be a collection of blood between the retina and the vitreous. The last is called a **preretinal hemorrhage,** and is situated at the macula as a rule, but is met with elsewhere in exceptional cases. Should the hyaloid membrane be ruptured the blood enters the vitreous.

Retinal hemorrhages are multiple in most cases, sometimes they are very numerous, but once in a while a single one is to be seen. If the eye has been wounded, or has received a contusion recently, the cause is plainly **traumatic** and the prognosis depends on the harm done to other parts of the eye, as well as to the retina. They

are met with after fracture of the skull, severe compressions of the thorax and abdomen, extensive burns of the surface of the body, and often are to be found in the eyes of infants just after birth. Ordinarily such hemorrhages as these disappear after a short time and do no noticeable harm, but sometimes they leave the vision impaired, and it is thought that congenital amblyopia can be traced in some cases to the injury inflicted on the sensitive elements of the retina by a hemorrhage that took place during labor, although it left no visible trace. Another form of traumatic retinal hemorrhage is seen when a small vessel ruptures during an operation for glaucoma, or for cataract, because of the sudden reduction in the tension of the eye. Other retinal hemorrhages are of **toxic** origin, when the patient is likely to give the history and to present the symptoms of chronic lead poisoning, suffocation with carbon dioxide, snake bite, poisoning with phosphorus, filix mas, potassium chlorate, or some such chemical compound as nitrobenzene, roburite, or hydracetin. Still others appear **spontaneously** in high myopia, glaucoma, and hæmophilia, but when these conditions are absent, and traumatism and poisoning have been excluded, they must be due to some disease that has altered the composition of the blood, or has produced changes in the walls of the vessels. Most commonly they form a symptom of retinitis. We meet with them in scurvy, purpura, and profound anæmia, though rarely in chlorosis. When we see a number of them about the papilla of an anæmic fundus it is well to ascertain whether the patient is not suffering from pernicious anæmia, whether other signs of retinitis are present or not. Retinal hemorrhages in women are caused sometimes by menstrual troubles, not only menorrhagia and metrorrhagia, but also amenorrhea and interrupted menstruation. Occasionally they occur during attacks of such infectious diseases as malaria, typhoid fever, influenza, and recurrent fever, in gout, in affections of the liver and spleen, and very often in nephritis, arteriosclerosis, and diabetes. Still other hemorrhages are caused by cardiac disease, and by obstructions to the general circulation.

Small White Spots

Small white spots commonly are multiple, and are to be found in any part of the fundus. We need to note whether they are accompanied by any signs of inflammation of the retina or of the optic nerve, and whether the vision is affected, for once in a while we see

a case in which they appear to be of no pathological importance. In such a case the vision is not affected, there are no accompanying signs of inflammatory trouble, and no alterations can be observed in their appearance after long intervals.

Gunn's Dots

A number of white spangles about the papilla in an otherwise normal eye were first described by *Gunn* as "crick dots." They may be met with in several members of the same family, and they seem to be simply a hereditary peculiarity.

Metallic Dots

Brilliant metallic looking dots are to be seen occasionally in both healthy and diseased eyes, but are of no importance. They are supposed to be caused by the total reflection of the light from minute irregularities in the surface of the retina.

Colloid Formations

Much more often we see a number of round or oval, pale, grayish yellow spots scattered about in the fundus, but tending to form groups in the macula and about the papilla. Sometimes they are confined almost wholly to the macula. They may be packed together pretty closely, but they do not coalesce. Here and there a retinal vessel may be seen to pass over them. When the vision is unaffected, the surrounding retina is normal, and there is no pigmentation, or other signs of trouble associated with them, these spots probably indicate the presence of colloid formations on the vitreous lamella of the choroid. If the vision is poor they may indicate a central choroiditis, a central retinitis, or a senile degeneration of the macula when they are grouped in this region. Elsewhere in the fundus we look upon such spots as symptomatic of retinitis when they are associated with other symptoms of this disease, otherwise as colloid formations. Colloid bodies may be met with at any age, but they seem to be more common in elderly people. They are rather larger than Gunn's dots, and are not common in the periphery of the fundus. The Germans call them "drusen."

Punctata Albescens

An extremely rare condition is one in which the periphery of the fundus is studded with a multitude of little white spots with no signs

of retinitis, no contraction of the visual fields, and no hemeralopia. This condition is supposed to be congenital, or to develop in early life, and has been observed in several members of the same family. This seems to have been the condition which was described by *Mooren* as retinitis punctata albescens, but this term has been applied also to a similar picture which is associated with the symptoms of pigmentary degeneration of the retina, of which it is thought to be an atypical form. Both conditions are rare, and are to be differentiated through a study of the vision and of the visual field.

Inflammatory White Spots

Most of the small white spots that are to be seen in the fundus indicate lesions in either the retina or the choroid. There is no rule applicable to all cases whereby we can determine in which of these membranes the lesions are situated, we have to take into account all of the conditions present, and to depend largely on a judgment trained by experience. Ordinarily a retinal spot is quite small, bright, sometimes brilliant, and is found about the center rather than in the periphery, while a fresh choroidal spot is apt to be larger, to have a yellowish, reddish yellow, or grayish tint, to be found frequently in the periphery, and to become pigmented when it grows old, yet a spot in the retina may be grayish, may be unusually large, or several may coalesce to form a single large one, and the situation is an unreliable guide. If a white spot covers a retinal vessel, or if it is accompanied by a hemorrhage, it is in the retina, while if it has accumulations of pigment in or about it, or if the markings of the choroid can be seen within it, it is in the choroid, but it often happens that such distinctive marks are absent, and both varieties are to be found in the same fundus.

THE RETINAL VESSELS

The retinal vessels furnish the field for much of our finest ophthalmoscopic work. We need to know whether they are normal or not in size, color, course, light streak, number, and walls, but such knowledge is not always to be obtained easily, because variations in all these respects are met with in normal eyes. Their apparent size is influenced by the same optical conditions which affect that of the papilla, a deeply situated vessel seems to be smaller than one

which is more superficial, and the actual sizes vary in different individuals. Doubtless many slight abnormalities escape the observation of the most expert, but if we utilize every opportunity to study the vessels, and to note the relations of slight changes to one another, as well as to other conditions, we shall gain an ability to detect deviations which ordinarily escape notice.

We observe first whether the arteries and veins ***maintain their normal proportionate breadth,*** for we are much more likely to notice such a change than to perceive an even broadening or narrowing of both sets of vessels. A disproportion in breadth is due in the majority of cases to a distention of the veins, which are darker and more tortuous than usual. The cause of the distention may be an impediment to the general venous circulation, such as accompanies cardiac disease, pulmonary emphysema, and pneumonia, or a local obstruction to the venous outflow, as in optic neuritis, choked disk, thrombosis of the veins, glaucoma, and the early stage of sclerosis. When the arteries are abnormally small while the veins are unusually large, the circulation in both is obstructed.

An **even narrowing** of both the arteries and the veins is one of the symptoms of anæmia of the retina, of pigmentary degeneration of the retina, and of a sclerosis of these vessels. An **even distention** of both the arteries and the veins is characteristic of hyperæmia of the retina, and of mild forms of retinitis. Both the arteries and the veins are distended in leucocythæmia, but the veins are enlarged to a disproportionate degree.

Sometimes a blood column that is plainly visible exhibits **irregular constrictions** here and there, which inform us that the walls of that vessel have undergone certain sclerotic changes, and we know that such changes are likely to be progressive.

We may see that certain vessels are bordered by **white stripes** which do not change when we move the mirror, and therefore are not light reflexes. The opacity revealed by these white stripes may overlay the blood columns and conceal them partially from view, as by a sheath, so it is convenient to refer to this condition as a **sheathing of the vessels.** In rare cases similar white lines appear congenitally in normal eyes and are to be differentiated from those of a pathological character by the total absence of other signs of sclerosis or perivasculitis after prolonged observation. In the great majority of cases such lines as these imply that an abnormal change has taken place in the walls of the vessel. If the sheathing is con-

fined to the neighborhood of the papilla we look for signs of a present or past optic neuritis, but in the periphery of the fundus it probably calls attention to a sclerosis beginning in the small vessels.

The **color** depends much on that of the background and on the situation of the vessels in the retina, as it is brighter when the latter are superficial, as well as when the background is dark, but it depends also on the condition of the blood. When the vessels are rather small, or not far from normal, except that they seem to be too light, and the fundus is pale, we think of anæmia, or chlorosis, perhaps of pernicious anæmia. Light colored, engorged vessels in a yellow fundus suggest leucocythæmia. Both arteries and veins are dark in cyanosis. The veins alone are very dark when there is an obstruction to the venous circulation, and they become almost black in thrombosis.

The vessels are tortuous normally, more so in hypermetropia, less so in myopia, but when they are distended the **tortuosity** is caused to increase, while a large number of little ones become visible. Contracted vessels tend to become straighter and less in apparent number. The most important point to observe concerning the tortuosity, at least in most cases, is whether any bend takes place in an anteroposterior direction, for such a bend is never normal. We may be able to recognize a slight curve of this nature only from a break in the light streak at the point where the vessel turns forward, or we may trace the vessel as it mounts the side of a high elevation by focussing successively on different parts of its course.

Although the **light streak** varies a good deal physiologically with regard to intensity, breadth, uniformity, appearance, and proportionate size on the arteries and veins, it affords considerable information as to the conditions of the vessels. It is broader when the blood pressure is low, narrower when it is high. Its absence suggests œdema, extravasation, or detachment of the retina, unless the blood column is very narrow, or the vessel is obliterated. Its disappearance at the beginning of a bend shows that the curve is either forward or backward. If it is particularly intense along a certain vessel we suspect commencing sclerosis, of which this is at times the first observable symptom.

The **walls** of the blood vessels are invisible normally, with the few exceptions which have been mentioned, and changes that have

taken place in them are made manifest by sheathing, or by irregularities in the diameters of the blood columns.

A little **swelling** attached to a vessel may be a sacculated, or a fusiform aneurysm, a varicosity, or an arteriovenous aneurysm. Such conditions are said to have been observed congenitally, but in most cases they have been caused by traumatism, syphilis, or sclerosis.

ANGIOID STREAKS IN THE RETINA

In rare cases a number of long, irregular, reddish brown, or nearly black streaks may be seen ramifying and anastomosing over the fundus and tapering outward beneath the blood vessels. The vision of the eye may or may not be perfect, but we are apt to see signs of choroiditis, especially in the periphery. These streaks may seem to extend outward from a grayish crescent near the papilla; retinal hemorrhages have been observed in connection with them, and, as a rule, they are present in both eyes. Their general appearance is that of obliterated blood vessels. The origin of these angioid streaks is not certain. Some observers think that they result from hemorrhages into the retina, while others are of the opinion that they are the remains of pigmented vessels, which were either congenital, or the result of inflammation.

STRIATE RETINITIS

In other rare cases we see white stripes, sometimes edged with black, that may be said in a general way to radiate in the fundus from the periphery toward the papilla, but hold this course very irregularly, present many branches, and sometimes have their ends teased out. They are to be differentiated from medullated nerve fibers and from proliferating retinitis by the fact that they lie beneath the retinal vessels; from direct ruptures of the choroid by the absence of a history of traumatism in which a force could have been applied to the back part of the eye; and from indirect ruptures by their position when the eye has suffered from a contusion. This is the so-called striate retinitis. It has been observed after the reattachment of a detached retina, and some writers consider it indicative of such a reattachment, but *Holden* among others inclines to the view that they originate in retinal hemorrhages. Vision is reduced commonly, but not always.

SCLEROSIS OF THE RETINAL VESSELS

In a young, or fairly young person sclerotic changes in the walls of the retinal vessels are strongly suggestive of **organic disease,** especially of syphilis, but unless they are accompanied by other symptoms of such a fundamental trouble it is hard to distinguish them from those induced by the degeneration commonly called senile arteriosclerosis, or better **angiosclerosis.** The changes in the fundus may be confined at first to the blood vessels, but later we are likely to find hemorrhages and white spots in addition.

During an ophthalmoscopic examination our attention may be attracted by the breadth, tortuosity, light color, and the intensity of the light streak of one of the **larger** vessels, which perhaps has a pulsatory movement, or by a great tortuosity of the **very small** vessels in a part of the periphery. Even though the eye is normal otherwise, and its functions perfect, we have good reason to fear that sclerosis has begun in that vessel or vessels, and we may be sure that such is the case if any sheathing is present.

In another case we may notice where an artery and a vein cross that the lower vessel seems to be **interrupted** for a greater distance than is apparent in other places where similar vessels of the same size pass over each other; a plausible explanation of this is that the walls of the upper vessel have lost a part of their translucency. The upper vessel may bend forward as it passes over the other, or it may be flattened; if the artery is above and the light streak stops at each margin of the vein to reappear as a bright dot midway between them, the artery makes a forward curve, while if the vein is above and its light streak broadens as it passes over the other vessel, we infer that the vein is flattened; in either case the walls of one or both of the vessels are abnormally rigid. If the vein is dilated on the peripheral side of the artery the pressure of the rigid walls of the latter has impeded the venous flow, and a hemorrhage is to be expected at this place sooner or later. All of these conditions are symptomatic of an early or somewhat advanced sclerosis.

A **sheathing** of the blood vessels, white bands along the sides of the blood column which may overlay it in places, is an almost indubitable sign of a perivasculitis associated with a thickening of the intima. It may be confined to a few of the small vessels in the periphery of the fundus, or affect certain ones throughout their

courses, but when it appears first on the vessels about the papilla it is commonly in connection with a neuroretinitis. If we look closely enough we can sometimes see minute white patches that look like scales on the arteries, which may otherwise appear to be either normal or diseased; these are considered by *Haab* to indicate syphilis. As the white bands of the sheathing grow broader the blood column may be reduced gradually to a slender red line, which once in a while we may have the opportunity to see break up into segments, and this finally is obliterated, so that the vessel is transformed into a white cord. During the course of this process little aneurysms and varicosities may form on the vessels and rupture.

Sometimes a vessel presents a **beaded appearance,** whether it is sheathed or not, and we may be able to see irregularities in the caliber of the blood column that are caused by encroachments on the lumen of the vessel by proliferations of the intima. This condition is known as **endarteritis** or **endophlebitis obliterans.** Occasionally we can see little glistening points beside the blood column which are caused by deposits of lime or cholesterin. The jutting irregularities produced by this proliferation of the intima afford excellent opportunities for the formation of thrombi, especially in the veins, and when they have brought the walls of the vessel sufficiently close together they interrupt the blood current, and cause signs of an occlusion suddenly to appear. The obstruction thus formed may yield enough to allow a flow of blood to resume its course, when the symptoms of occlusion will improve, or pass away for a time, but we must expect the proliferation to continue until the circulation is completely and permanently blocked.

The **cause** of the sclerosis must be learned from an investigation of the organism, but occasionally a hint as to its nature may be obtained from the ophthalmoscopic picture. If the arteries are sheathed much more than the veins we strongly suspect syphilis, particularly if the patient is comparatively young, though such a sheathing may be due to renal disease. In an elderly person who gives no marked symptoms of any organic disease we refer the sclerosis to a senile degeneration of the walls of the vessels, provided that all known tests show his kidneys to be functionating normally, but if his urine has a high specific gravity and contains an excess of uric acid with some albumin but no casts, while the phenolsulphonephthalein and McLean index tests give negative results with

regard to renal trouble, we must take into account the possibility of gout, especially when there is an accompanying retinitis.

LARGE WHITE SPOTS IN THE FUNDUS

A large white, or light colored spot, that does not change its shape or place with movements of the head or mirror, indicates an abnormal condition of the retina, of the choroid, or of both. It must be caused either by an opacity of the retina, or by a defect in the choroid, which usually includes the retina, that allows the sclera to be seen. It may stand out clearly and be accompanied by no other defects, or it may exhibit other changes as well. It may be hazy, the retinal vessels associated with it may present various alterations, sometimes showing elevations or depressions of the surface, it may have patches of pigment in or about it, the marking of the choroid may be visible within its area, and it may be sharply defined, or its margins may shade off into the adjoining parts of the fundus. It may be round, crescentic, oval, parabolic, triangular, or irregular in shape, and it may be congenital, or acquired either from the stretching of the tissues, or from disease. The bearing of all of these various conditions upon the diagnosis will be brought out best by a study of the different congenital and morbid cases in which they appear.

Medullated Nerve Fibers

Occasionally a portion of the papilla and of its margin is hidden by a striated opacity with indistinct borders, which varies in color from bluish white to reddish yellow, and terminates in flame shaped processes in the retina, while the rest of the disk looks redder than normal through contrast. It conceals the deep vessels of the retina, but the superficial ones may stand out in it sharply. These characteristics mark a large white spot as a patch of opaque or medullated nerve fibers, which is normally present in the retinæ of some animals, like the rabbit, but is not common in man.

Although the optic nerve fibers lose their medullary sheaths at the lamina cribrosa, it happens sometimes that a larger or smaller number regain them after passing through the lamina, and retain them for variable distances. If such fibers are sufficiently numerous to reflect all of the light, the spot appears to be glistening white,

but a thin layer is more or less translucent, so as to appear yellowish, or reddish yellow.

These fibers may form a flame shaped ring about the papilla which resembles somewhat those seen sometimes in neuroretinitis, choked disk, and sclerosis of the choroid, but when the visible blood vessels are not swollen, there are no hemorrhages, the opacity crosses the margin of the disk, its surface is destitute of choroidal markings and is seen to be finely striated when we have the correct focus, all of these conditions are excluded. It is possible for a proliferating retinitis to resemble medullated nerve fibers, as it presents no typical arrangement of its connective tissue, but as this growth of tissue always starts from a hemorrhage, or from a laceration of the retina or of the choroid, it is excluded when there is no history of a serious lesion of the eye, and the rest of the fundus is normal.

Sclerosis of the Choroid

When we see a circumscribed spot of almost any shape and size that contains a network of yellowish bands separated by pigment, and has margins which are indented by narrow projections, we recognize this to be an area in which the choroidal vessels are sclerotic and the pigment epithelium of the retina has been destroyed. Some of the yellow bands may have narrow red lines along the middle, from which we conclude that these vessels are not quite occluded. In other cases the bands are white, when we infer that the vessels have atrophied so that we see their former situations marked out on the sclera by the intervascular pigment. In still other cases the latter has disappeared and we see nothing but the white sclera with lumps of pigment, or black spots, in or at the margin of the white spot, which they may or may not define. Such spots may be **congenital,** as in coloboma of the choroid, or of the macula, in inferior conus, and in some cases of ordinary conus, or they may be **acquired** from a stretching of the membranes, as in posterior staphyloma, or from disease like choroiditis, so we have to take into account the age of the patient, the history, the refraction, the situation of the defect, the presence or absence of other defects in the fundus, and the bodily health before we decide whether a given white spot of this nature is congenital or acquired. If it is not congenital we need to learn the cause of the sclerosis.

Defects about the Papilla and the Macula Lutea

A circle about the papilla, not necessarily complete, which is fairly even in width and has an indistinct outer margin, is called a **halo.** A more or less narrow circle about the papilla that is broadest on its temporal side, or more commonly a crescent of varying width that is applied to the temporal margin of the papilla, and has its outer edge sharply defined by a greater or less accumulation of pigment, is a **conus** unless it contains choroidal markings, when it may be a **posterior staphyloma,** or an area of sclerosis of the choroid. A similar crescent at the lower margin of the papilla is an **inferior conus.** A large defect in the lower part of the fundus with its long diameter vertical and its floor on a lower plane than the retina, is a **coloboma of the choroid,** which may or may not be associated with a **coloboma of the sheath of the optic nerve.** A very large, round or elliptical, depressed spot with a regular margin in the macula probably is a **coloboma.** All of these have to be differentiated from white spots that indicate pathological lesions of the choroid or retina.

The Halo of the Papilla

This is a more or less broad, complete or partial circle of a grayish, yellowish, or reddish color, immediately adjacent to the papilla, which fades away into the surrounding retina with no distinct margin. The patient is elderly in nearly every case, and the halo may be due to senile, or to glaucomatous changes in the retina about the nerve head. If any of the blood vessels hook over the margin of the papilla the halo is **glaucomatous,** and symptoms of a far advanced glaucoma will be found in the eye; otherwise it is **senile** and has been caused by an atrophy of the choroid and of the pigment epithelium of the retina. The halo of the macula, which is to be seen in young people and has been described under the light reflexes of the fundus, is altogether different and of no pathological importance.

Conus

When the papilla presents on its temporal side a whitish crescent, or is surrounded by a ring that is broadest temporally, and this crescent or ring has a pigmented edge toward the retina, so that the general appearance is as though the pigment ring had been

pulled away from the scleral ring so as to leave some of the sclera exposed, we see what is called a conus. The choroid and the pigment epithelium of the retina over this area may be absent, or it may be rudimentary, so the **color** of the conus varies. It may be white at the margin of the disk, become progressively yellowish, reddish yellow, and reddish brown toward its pigmented margin, or it may be entirely of any of these colors. Often it is mottled irregularly. A similar crescent is sometimes, though rarely, seen on the nasal, or the upper margin of the papilla, but when we see one below we have to deal with a different condition, that of an inferior conus.

We are justified in pronouncing **congenital** a conus that is found in a healthy eye which is not myopic, and shows no tendency toward myopia, for in such a case the probability is that it is nothing more than an unusually broad scleral ring. It may present a superficial elevation over which the vessels curve, or it may encroach on the scleral canal so as to flatten a portion of the margin of the disk, but such modifications are not common.

A conus may be of the same nature in an apparently healthy, moderately myopic eye, but that it is not in the majority of cases is evident from the fact that it is present in several times as many myopes at the age of twenty as at that of ten. An **acquired** conus is thought by some to be a definite sign of school myopia, that is, of a myopia which is the result of a continuation of the normal elongation of a child's eye beyond the point at which it should cease, that is induced by the conditions attendant upon school life. A positive statement whether a given conus is congenital or acquired cannot be made from its appearance alone, though as a general thing the acquired is the larger, and perhaps the whiter, but sometimes it is made possible from a foreshortening of the papilla.

Retinal vessels may pass over a conus, but an enlargement of the blind spot proves that the retina is not present in its entirety, aside from the absence of the pigment epithelium, as it has lost its function. Except for the foreshortening mentioned the papilla usually appears to be normal, though it may be a little indistinct, or reddened by contrast, perhaps by the foreshortening of its vessels.

Posterior Staphyloma

It is not right to say that the term conus should not be applied to the crescent found in myopia, or that a conus and a posterior

staphyloma are the same,—the two conditions differ anatomically and in prognosis, though it is true that we are not able to differentiate them ophthalmoscopically in every case. A posterior staphyloma is a localized outward bulging of the sclera, usually about the papilla. In the majority of cases it is on the temporal side, so that the papilla is in the nasal wall of its cavity, but sometimes it surrounds the entrance of the nerve and contains the papilla in its floor. It is seldom if ever met with except in high myopia, it is symptomatic of the progressive or malignant variety, and some believe that it indicates a congenital predisposition to this disease.

The measurement of differences of level is not as easy in high myopia as in other refractive conditions of the eye, and is unreliable for the diagnosis of posterior staphyloma, as a rule. The crescent usually is broader than that of a conus, and sometimes we see in it gray or reddish gray curved lines, that impart a sort of terraced appearance when two or more are present, which are shadows cast by the margins of the staphyloma, and are diagnostic whenever they are seen. Ordinarily they appear only to the nasal side of the papilla, though occasionally they occur to the temporal side. These are the **Weiss-Otto shadow rings.** They must not be confounded with the Weiss reflex ring, a bright curved line to be seen in the same situation in some young persons, which is caused by a reflection of light from the retina.

The optic nerve enters the eye to the nasal side of the posterior pole, and presents its disk full face to our line of vision when the curvature of the posterior pole and the walls of the canal through which the nerve enters are normal. When the curvature is changed by a stretching of the sclera at the posterior pole the nerve enters at an angle to our line of vision, so that we see the papilla in partial profile, or foreshortened. At the same time the dragging on the temporal wall of the canal in the sclera through which the nerve enters may have rendered it more or less parallel to the nasal, a change that may be represented diagrammatically as from /.....\ to /...../, and then the choroid and retina may overhang the nasal margin, while they seem to be pulled away from the temporal. This is the condition called **supertraction,** which is thought not to be present congenitally.

As a rule, the papilla is **foreshortened** to a greater degree than in conus, and forms a vertical ellipse if the staphyloma is strictly on its temporal side, or an oblique one if the latter extends downward

or upward as well as outward. It can be circular only when it forms the floor of a depression which is equally deep on all of its sides.

These symptoms are present only when the staphyloma is large, deep, and far advanced, so we need others by which to differentiate it in its early stage from conus. *Adam* makes the statement, with some reservation, that the presence of sclerotic choroidal vessels in the crescent marks it as a posterior staphyloma, while their absence goes to show it to be a conus. If we happen to see in a case of myopia what looks like an ordinary conus, except that the choroidal vessels are visible within it, some perhaps obliterated while others may be full of blood, and reexamine it from time to time, we may be able to see this portion of the choroid pass through the stages of atrophy while the staphyloma grows larger and deeper, and the myopia increases. If this is correct the presence or absence of the choroidal markings in what appears to be a myopic conus is a diagnostic symptom of much value. The only other condition that would need to be differentiated would be a sclerosis of the choroid from some other cause, and this is not likely to be associated with a progressive myopia.

Inferior Conus

A white crescent that embraces the lower margin of the papilla is regarded generally as a **rudimentary coloboma of the choroid.** Dark spots may be scattered about in it, but the markings of the choroid cannot be traced. The papilla is apt to be small, and obliquely or transversely elliptical, and often the line which divides it from the conus is far from distinct. It is apt also to be peculiar and anomalous both in its excavation, and in the distribution of its vessels. The fundus almost invariably is albinotic in type in its lower part, even though it may be of a uniform red elsewhere. The eye is amblyopic and highly astigmatic, as a rule. It is possible for a white spot or crescent at the lower margin of the papilla to be due to disease, but in such a case these peculiarities of the papilla and fundus will not be found to be present.

Coloboma of the Choroid

Once in a while, usually though not always in connection with a coloboma of the iris, a brilliant white area is to be seen in the lower part of the fundus, which has a sharply defined, pigmented border,

and is on a lower level than the retina. It may have the form of an ellipse, with its long axis vertical, lying between the papilla and the periphery, or it may have about the shape of a parabola with its vertex above, at, or below the papilla, its base lost in the periphery. This is a coloboma of the choroid, a congenital defect supposed to be due to a faulty closure of the fetal cleft. Occasionally it is visible only when the patient looks far down, because it is so completely peripheral.

Although the general **color** is a shining, pearly white, portions of the coloboma may be pinkish, bluish, greenish, or gray, while brown or black spots are to be seen within it. The floor is quite uneven, as is shown by the play of light with the movements of the mirror, and the tortuous courses of the vessels that pass over it.

The **papilla** is normal in most cases when it is not included in the coloboma. Its lower **vessels** may run freely over the surface of the latter on a level with the rest of the retina, and in such cases they can be shown by parallactic movements to be elevated above the floor of the defect, but more often the larger ones either turn aside at the margin of the coloboma and send small branches to ramify over its surface, or plunge over the margin with a distinct bend, and then wind about on a level plainly lower than that of the retina. It is interesting to note in this connection that although a defect usually exists in the visual field that corresponds to the coloboma, it is not always present. A connection between the preservation of vision in this area and the maintenance of the retinal vessels at the level of the retina can hardly be said to have been demonstrated unquestionably as yet, but it is certain that a coloboma of the choroid is not accompanied necessarily by a corresponding defect in the retina, even though the two coexist in many cases.

When the papilla is partially or wholly included in the coloboma we may find some difficulty in discerning its outline, but this can be distinguished usually. It is horizontally or obliquely elliptical in most cases, and the distribution of its vessels is abnormal.

The characteristics manifested by the general appearance of the defect, by the retinal vessels, and in some cases by the papilla, serve to differentiate a congenital coloboma of the choroid from a white spot of similar size, shape, and position that might have been produced by disease. In addition to this we are likely to find other malformations in the eye, for such a coloboma seldom occurs as the only congenital defect.

Coloboma of the Macula

A large, roundish, or transversely elliptical, white, or yellowish spot in the macula, which is depressed below the surface of the surrounding retina, is traversed by retinal vessels, sometimes contains a network of pigment, and has a regular, pigmented margin, possibly may be a coloboma of the macula, but as very few are on record all other white spots must be carefully excluded. The defect is said to be confined to one eye, as a rule, but occasionally to be bilateral. The vision may range from normal to a high degree of amblyopia, but when it is good a scotoma is apt to be present.

The conditions of which we must think when we see a large white spot in the macula are traumatic œdema of the retina, a coalescence of small white spots that are symptomatic of retinitis, circinate degeneration, tuberculosis, proliferating retinitis, occlusion of the central artery, amaurotic family idiocy if the patient is a child, a cyst, and central choroiditis, or sclerosis of the choroid. Most of these are excluded at once by the appearance and the history. A contusion of the eye followed by a dense grayish opacity of the retina, with practically normal vessels, that passed away in a few days, produced a traumatic œdema. An irregular whitish mass with no pigmentation may be resolved into a coalescence of small white spots, but usually they are associated with well marked signs of a neuroretinitis; when such signs are absent it may indicate circinate degeneration if the patient is elderly, or a tubercle in a child, but in all of these the regularity of the margins of a coloboma is lacking. The white mass of a proliferating retinitis is irregular, lies above the level of the retina, and is accompanied by signs of the preceding hemorrhage or inflammation. Both occlusion of the central artery and amaurotic family idiocy are marked by a red spot in the fovea; in the former there has been a sudden loss of vision, in the latter the child is growing more and more lethargic. A cyst is elevated, has no pigmented margin, and has a translucent appearance. The only conditions really productive of doubt are a central choroiditis, and a far advanced stage of sclerosis of the choroid in the macula. The history is very important to the differentiation, for one of failing vision, or of sudden blindness, excludes coloboma at once. If the eye is myopic, if it exhibits other changes that may be ascribed to traumatism, or to disease, and if the patient is elderly, syphilitic, tuberculous, or seriously ill with such a disease as diabetes, a pig-

mented white spot in the macula probably marks a central choroiditis, and does so with certainty if the vision of the eye has been failing. A far advanced sclerosis of the choroid may be congenital, but such a condition is as uncommon as a coloboma, so we must be careful in asserting any distinctive marks to be in favor of either, and can be certain of only a few points. Coloboma is stationary, while sclerosis is progressive, and it is possible to make the differentiation from accurate measurements made during a prolonged period of observation. When an adult is known to have had such a defect from early childhood and the eye to have kept the same vision since that time, whether it is good or amblyopic, the diagnosis of coloboma is practically positive. When the history is unknown and the patient has good vision, we may be pretty sure that such a large white spot in the macula is a coloboma, but when his vision is poor, the diagnosis is doubtful. We notice in such a case the margins and the markings of the floor of the spot, for an indented border without pigment and with visible sclerotic choroidal vessels would lead us to a diagnosis of sclerosis, while a regular, pigmented border with no definite choroidal markings in the floor would favor that of coloboma. Other points that would be favorable to a diagnosis of the latter are youth of the patient, and absence of any general disease that might cause sclerosis.

Rupture of the Choroid

When we see a long, narrow, white or yellowish, vertical crescent with sharply defined pigmented edges, across which the retinal vessels run, near the posterior pole of the fundus and concentric with the margin of the papilla, we infer that at some time the eye received a blow that produced a rupture of the choroid by contrecoup. Two or more ruptures may be nearly parallel. Usually they are of different sizes and sometimes their margins are more or less jagged. It is very uncommon for us to see a rupture of the choroid on the nasal side of the papilla, though it has been known to occur, and another rare condition is such a horizontal rupture as has been portrayed by *Lawson.*

A rupture of the choroid may be hidden by a hemorrhage immediately after the traumatism, but it stands out plainly when the blood has been absorbed. At first it may be yellowish, especially at the ends, but in most cases it becomes pure white later. Little red

spots are visible in it at times, and may be ascribed to minute hemorrhages, or to tufts of little vessels. The lesion is almost always permanent, though very small ruptures are said to have been known to heal.

Irregular lacerations of the choroid that form long white stripes in the periphery are not as common as indirect ruptures, but are to be seen occasionally as the direct result of a blow on that part of the eye. These white stripes differ from those seen in striate retinitis in that they do not radiate toward the disk, as a rule, and are always the result of a severe traumatism.

The vision of the eye may be badly impaired, or scarcely affected by the rupture of the choroid, according to the situation of the lesion. If it passes through the macula, or between this and the papilla, as it very often does, the sight is injured. The retina is torn in rare cases, and cicatricial changes may take place which will cause the vision to deteriorate later.

Proliferating Retinitis

A portion of the fundus may be hidden by white or bluish white masses or bands of tissue that rise from the retina into the vitreous. Sometimes these interlace, and allow us to catch glimpses of the papilla, or of some other part of the fundus, through the interstices. The white color is somewhat duller than that of medullated nerve fibers, which sometimes have to be excluded as the bands may have a similar striation. This exclusion is made by observing whether the bands show a parallactic movement which locates them as above the level of the retina. Striate retinitis, in which the white stripes lie beneath the retinal vessels, is excluded at the same time. Occasionally the bands or masses are vascular, but the vessels are newly formed and can be distinguished readily from those of the retina, as a rule. This condition is called proliferating retinitis, though it is not a retinitis at all. It is a **proliferation of the connective tissue** of the retina into the vitreous. Almost invariably the patient gives a history of repeated large hemorrhages into the vitreous, less often one of a laceration of the retina or choroid, but a similar condition is said to have been met with congenitally. The masses of tissue have no typical arrangement, though they are apt to follow the courses of the blood vessels, as in the majority of cases they start from them, or from their adventitial sheaths. Sometimes blood is

present in the vitreous, and detachment of the retina is not an uncommon accompaniment.

The causes are those of the hemorrhages, which most commonly are of the recurrent juvenile variety, or are caused by syphilis, arteriosclerosis, or diabetes. The prognosis is bad. The proliferation is likely to continue, and the contraction of the newly formed connective tissue to detach the retina.

Œdema of the Retina

The appearance presented by an œdema of the retina varies from that of a slight, superficial haze, to that of a whitish gray cloud, and the ease with which it can be perceived depends mainly on its density. A very slight degree can be recognized only by care in noticing the distinctness with which all parts of the fundus are visible, except when the fundus is of the tessellated or albinotic variety, when a part of the choroidal markings is obscured. A slight œdema is particularly hard to detect at the macula, unless it is associated with little white spots. When a patient complains of a rapid, great deterioration of vision, for which there appears to be no adequate explanation, we should consider carefully whether the stippling of the macula is as plain as usual, and we should test his central vision with a small blue disk. If the stippling is not quite plain, and particularly if blue appears to be green, the presence of œdema at the macula is very probable. When only one eye is affected a comparison of the two maculæ is a great help. Occasionally we can make out a few very minute, almost linear, grayish, or yellowish marks that decide the question. This faint haze, perhaps with these almost imperceptible marks, may be the only ophthalmoscopic symptom of a **central retinitis.**

A vague haze about the papilla almost always indicates a slight œdema characteristic of retinitis, even when it is not associated with visible changes in the vessels, or hemorrhages, which are apt to be present when the œdema is more marked. As a rule, œdema of the retina is sufficiently distinct to be quite noticeable as a more or less dense cloudiness, which may be present almost anywhere in the fundus. Occasionally when such an œdema forms a ring about a normal or hyperæmic papilla, and is densest at a little distance from the margin of the latter, it is of diagnostic value, particularly when it appears in a patient who is suffering from a purulent otitis media,

as it causes us to fear an intracranial extension of the inflammation, of which this may be the first sign.

Traumatic Œdema of the Retina

When an eye has been blinded by a contusion we may see at its posterior pole a more or less dense, grayish opacity, elevated slightly if at all, in which the vessels of the retina stand out plainly and may be slightly dilated, but are not abnormally tortuous. The papilla may or may not be hyperæmic, and other results of the traumatism may or may not be present in the eye, though a traumatic myosis is a frequent accompaniment. This is a traumatic œdema of the retina, also called **Berlin's opacity.**

A traumatic œdema covers the macula in most cases, but occasionally it does not, and then its effect on the vision is not so extreme. It may vary in color from a reddish gray, perhaps with a dark center that makes the discoloration look like a ring, to gray or yellowish white, which is fairly uniform, or densest in the center. It reaches its maximum in about twenty-four hours, then begins to pass off, and usually is gone in three or four days. The prognosis depends on the harm that may have been done otherwise, for the œdema passes away completely, vision returns, and commonly no permanent ill effects are experienced, though occasionally a discolored spot in the macula and a central scotoma remain.

HOLE IN THE MACULA

Sometimes the vision of a patient does not return satisfactorily after the immediate effects of a traumatism have passed off because of a central scotoma, which may be relative and slight, or absolute. When the scotoma is absolute and large we may find a round or oval red spot in the macula, about half as large as the papilla, through which the choroidal markings are visible, but with no pigmentation. In such a case we say that the patient has a hole in his macula. This red area can be made out to be depressed in most cases, about half a millimeter, as a rule, according to *Ogilvie.* The surrounding retina may or may not be detached. It is hardly necessary to say that the damage to the vision is permanent.

OCCLUSION OF THE CENTRAL ARTERY OF THE RETINA

When a patient has suffered a sudden, total loss of vision in one eye, which may have been preceded by one or more similar attacks at intervals of weeks or months, or may have been wholly unexpected, and we find a large area of the fundus that includes both the papilla and the macula to be grayish white and opaque, the papilla red with its margins hidden, the arteries very small or invisible, the veins not far from normal, but not pulsating when pressure is made on the eyeball, and a cherry red spot at the site of the fovea, there is no question that the central artery has been occluded. The only other condition in which we find a cherry red spot at the fovea is amaurotic family idiocy, in which the patient is very young, the retinal opacity does not reach the papilla, and the vessels are normal.

After a few days the papilla is no longer red, its margins are again sharply defined, the whiteness of the fundus is fading, the cherry red spot is no longer conspicuous, and the arteries have refilled. Occasionally vision has returned, but more often it has gone forever, and in the absence of the history we may be at a loss for the diagnosis. Usually we can find some indications of arteriosclerosis, which will guide our suspicions and, with the history of an onset of sudden blindness, suffice to tell us what has happened. After many years have passed we can still make the diagnosis from the history taken in connection with a white atrophic papilla of normal level with threadlike arteries, a fine grayish pigmentation of the retina near the papilla, and a circle of fine, bright yellow or reddish spots about the fovea, usually having a slight pigmentation, which is called a **coronula.** Sometimes the main artery is apparently sheathed at its exit from the nerve head.

The opacity of the retina is due to a rapid **necrosis of its inner layers,** together with an œdema. Ordinarily it is of a pale gray color, rarely has a bluish or greenish tint, and sometimes is of a milky or yellowish white. These inner layers are absent at the fovea, the dark color of which consequently is allowed to stand out in marked contrast to its pale surroundings and to give rise to the characteristic **cherry red spot.** Occasionally this red spot may look a little hazy, and *Loring* quotes *Gowers* as reporting a case in which it was totally obscured. When they are partially empty the arteries are often bordered by delicate white lines. Sometimes they appear

to be better filled in the periphery of the fundus than near the disk, sometimes they are very narrow in the middle though moderately or normally filled at both ends. The veins are normal or a little distended, as a rule, but they may be contracted near the papilla. Small retinal hemorrhages often are present. In the majority of cases the ischæmia of the retina is apparent soon after the attack, perhaps in twenty minutes, but it may not come on for a day or more, and it is said to have been delayed two weeks in one case. In very rare cases the retinal hemorrhages are so great as to simulate a thrombosis of the central vein, and then the differentiation can hardly be made clinically.

Exceptionally we find on the temporal side of the papilla an **area** of the retina that is of its normal color, and in which the vision has been preserved, **supplied by cilioretinal vessels** that spring from the temporal margin of the disk to nourish this area. The size of the area depends on that of the cilioretinal vessels, and when these are large enough to supply the macula the central vision may be preserved, but such a case is extremely rare.

If we happen to be looking at the fundus just as the blood current has almost stopped, or as it recommences, we may have the opportunity to see dark red cylinders separated by clear spaces pass through one or more of these vessels; this is called the **granular current.** Usually it appears first in the veins, but it is to be seen in the arteries also. Occasionally the direction of the current is reversed, or we may see the cylinders oscillate back and forth.

For many years the cause of this occlusion was supposed to be embolism, and so it probably is when the arrest of the circulation occurs in some cases of heart disease, after operations on the nose, or after injections of paraffine beneath the skin of the face. *Loring* was the first to question the dictum of embolism seriously, and to advance the belief that the occlusion was due in many cases to a disease of the vessel itself. This view has gained ground slowly but steadily, until at the present time the great majority of cases are ascribed to **endarteritis obliterans.** A few cases may be caused by thrombosis of the artery. Among the rare causes of occlusion are traumatism, violent compressions of the thorax and neck, and pressure on the optic nerve.

Occlusion of a Branch of the Central Artery

When we see a triangular spot of ischæmia in the fundus, with its apex at the papilla, which is supplied by small, straight vessels, and corresponds to a triangular, absolute defect in the visual field, we know that the arterial branch which normally supplies this region has been occluded. If the area includes the macula we shall see a cherry red spot and find the central vision abolished, but if it does not there will be no red spot and central vision will be present. We may or may not be able to see a point at which the artery suddenly becomes very small, or is changed to a white cord.

The differentiation of this condition from a large white spot caused by some other lesion is made through the narrowness and straightness of the vessels, together with its triangular form, and the absolute character of the defect in the field.

RETINITIS

When a patient whose eyes outwardly appear to be normal tells us that he seems to see through a mist, that he needs a strong light to enable him to examine anything closely, or that his vision is still more impaired, and we find in his retina œdema, enlarged vessels, white spots, and hemorrhages in some degree or combination, we say that he has a retinitis. All of these symptoms may be present, and they may be associated with an optic neuritis, or a choroiditis, or one or more of them may be absent, and they may be of any degree from a faint, scarcely visible haze about the fovea with no other perceptible changes, to much œdema, enormously swollen and tortuous vessels, and many white spots and hemorrhages, associated probably with a well marked optic neuritis. The changes may be confined to the region of the macula, when the retinitis is called central, but more often they are generalized, with a tendency to group about the papilla and the macula. When the hemorrhages form the most prominent symptom we call the retinitis hemorrhagic.

We can hardly conceive of an inflammation which involves the nerve fibers of the retina and leaves the optic nerve unaffected, or of one that destroys the pigment epithelium without attacking the choroid. The fact is that we can detect evidence of an optic neuritis, or of a choroiditis, in the great majority of cases, while sometimes all three tissues—retina, choroid, and optic nerve—are affected.

It is also true that a lesion in the choroid can manifest itself only through changes produced in the retina. Consequently nearly every case of retinitis may be termed properly a neuroretinitis, or a chorioretinitis, but we are accustomed to restrict the use of these terms so that they will denote conditions in which the symptoms referable to the optic nerve and the retina in the one case, and to the choroid and the retina in the other, are about equal. When the changes in the retina predominate, and those in the papilla or the choroid are subordinate or secondary, we speak simply of a retinitis.

The effect which is produced on the **vision** varies with the intensity of the inflammation, but to a greater degree with its situation. The sight may remain very good so long as the fovea is not affected, but the slightest lesion in this place produces a great impairment.

Retinitis is not a disease per se, but is symptomatic of some other trouble, most often of a general disease; so our diagnosis is not complete until its **cause** has been ascertained. Sometimes the patient is known to be suffering from a certain malady before the examination is made, but often this is not the case. Occasionally we are able to get a hint from the details of the picture that arouses our suspicions of a certain ætiology, but these details are never sufficiently reliable to be depended on alone, we have to take into account all of the symptoms presented by the entire organism, and to avail ourselves of all clinical and biological methods of differentiating disease. A slight haziness of the retina outside of the macula may pass unnoticed, or we may have grave doubts as to its existence, unless the fundus happens to be of the tessellated or albinotic type and the markings of the choroid are locally obscured, but when the œdema is sufficient to make the retina gray we have no difficulty in seeing the opacity. Such a condition may be ascribed to traumatism, if the eye has received a contusion recently, or has been exposed to bright light reflected from snow or water, or to such a flash of light as is produced by an electric short circuit, but in the absence of any of these causes we think at once of some systemic disease, first of all of syphilis and of nephritis. After the cause has been determined we indicate it by an adjective qualifying the word retinitis, so as to give a complete diagnosis. In this way we obtain a large variety of forms, only a few of which have characteristics that even approach being distinctive.

Once in a while the examination of the fundus is of importance

in ***deciding the diagnosis of a general disease*** after it has been narrowed down to three or four by exclusion. For example, we may suppose that a patient with high fever and stupor presents symptoms which are about equally indicative of sepsis, meningitis, miliary tuberculosis, and typhoid fever, while all other diseases have been excluded. If such a patient has inflammatory changes in his retinæ, we can rule the last out, at least with a high degree of probability, especially in the absence of a distinctly positive Widal reaction, for retinitis is extremely rare in typhoid fever. The opposite does not hold true, for there is no disease in which retinitis is a necessary accompaniment, and a normal fundus can furnish no aid in making the diagnosis. But when changes are present they give a great deal of help, for if there is an optic neuritis it decides the question in favor of meningitis, and if it is associated with a few rather large, yellowish spots scattered over the fundus with no hemorrhages, we say that the meningitis is tuberculous. If the fundus presents similar spots with no changes in the papilla or the vessels, the diagnosis is miliary tuberculosis. If little white spots are to be seen congregated about the papilla with little or no reference to the vessels and are associated with hemorrhages, the patient is suffering from sepsis. Still the fact must be emphasized that if it were not for the concomitant general symptoms, and the previous exclusion of all other diseases, none of these ophthalmoscopic pictures could be considered diagnostic.

Central Retinitis

When the vision is reduced considerably and we can find no explanation for the reduction in the refraction, the media, the papilla, or apparently in the retina, we should dilate the pupil and investigate the macula. This is particularly important when the failure of vision occurs during pregnancy, or after an attack of some acute infectious disease. We may detect an indefinite haze, so slight that we hesitate to say that it is there, perhaps with a few indistinct dots about the fovea, the existence of which are questionable, or the haze and spots may be quite evident. If the eye is myopic we may decide that this is the first sign of a central myopic choroiditis, if the patient is elderly we may conclude that senile degeneration of the macula has begun, but we should make a systematic physical examination in every case to detect or exclude the diseases of which it may be a symptom.

The difficulties that are apt to attend diagnosis in this class of cases are illustrated by two examples taken from my private practice. A boy 7 years old, who was convalescent from scarlet fever, had a vision of 20/70 in each eye which was not improved by glasses. Under atropine his total hypermetropia was 1.5 diopters without astigmatism. Both maculæ seemed to me to be a trifle hazy, with faint, minute, yellowish specks about the foveæ which resembled a rather coarse stippling, but nothing of the sort could be seen by two eminent ophthalmologists who saw the boy in consultation, and agreed that he had congenital amblyopia in both eyes, for which nothing could be done. Three months later the haze and specks had disappeared, the vision was 20/20, and later rose to 20/10. Subsequently the boy developed symptoms of a chronic nephritis. The second case is that of a lady about 30 years of age who had nothing to account for a reduction of vision to 20/40 except a few white spots in her right fundus, and an indefinite haze in the center of her left macula. Three examinations of her urine gave negative results, but on the fourth albumin and casts were found. This case illustrates also the importance of not relying on a single examination of the urine to exclude nephritis in any case of retinitis of unknown origin. Repeated examinations often are necessary, and it is much better to subject the patient to the modern renal tests to determine the functionating power of the kidneys than to rely wholly on the urine.

The **differentiation** of retinitis as such is easy, as a rule. Macular and maculocerebral degenerations appear at definite ages of childhood, are marked by specks of pigment, and the latter is associated with a slowly developing imbecility. The presence of pigment may suffice to mark a central choroiditis, or a senile degeneration of the macula, though perhaps not in all cases; sometimes it is necessary to wait and to watch for the appearance of pigment, or of additional symptoms of inflammation. If the lesion passes away spontaneously we may say that the diagnosis is certain. A condition characterized by a number of pale yellowish dots in the macula, described by *Tay* as a choroiditis, may be indistinguishable except by its course, but it is possible that this is simply a variety of what we consider a central retinitis. When we see in the macula a number of round or oval, discrete, yellowish spots, which show no tendency to fuse, and there is no other sign of retinal trouble, we test the vision, for if this is not impaired we know them to be Gunn's

dots if the patient is young, or colloid formations on the vitreous lamella of the choroid, if he is old, and in either case to be not indicative of disease.

Septic Retinitis

When a retinitis develops in a patient who is suffering from septicæmia, or from some other form of sepsis, we call it septic. Vision is said to be good when the patient is in such a condition that it can be tested, but this seldom is the case. The most marked feature in the majority of cases is the presence of white spots about an uninflamed papilla, with hemorrhages from the large vessels, which otherwise appear to be unchanged. Optic neuritis occurs rarely, the vessels usually are about normal in size, the hemorrhages sometimes are preretinal, while the white spots vary in number; sometimes they are numerous, sometimes few, and occasionally they have been known to be absent.

Metastatic choroiditis occurs under the same conditions, and its primary symptoms may resemble those of septic retinitis closely at first, but the diseases can be differentiated very soon. Septic retinitis runs a slow course with no inflammatory symptoms or external signs, beyond possibly a subconjunctival hemorrhage, and may end in recovery if the patient gets well, while in metastatic choroiditis inflammation develops quickly, renders the eye blind, and usually runs a rapid course to terminate in panophthalmitis. Nevertheless the two diseases are closely related and it is possible for septic retinitis to attack one eye while metastatic choroiditis attacks the other.

Albuminuric Retinitis

A snap diagnosis of albuminuric retinitis often proves correct, as nephritis is the disease that most commonly excites an inflammation of the retina, but frequently it is incorrect, and it is well for us to be acquainted with the signs that render this or some other diagnosis to be the more probable.

When we find an optic neuritis of any degree from a hyperæmia to a choked disk, an œdema of the retina that is densest about the papilla and contains at some little distance from the margin of the latter irregularly arranged, cottony white spots that cover the retinal vessels and look somewhat like fleecy white clouds, or when we see

much smaller white spots in the macula, or grouped along the vessels, with hemorrhages of various sizes and shapes lying scattered about, the probability that the retinitis is albuminuric in character is strong. The white spots in the macula may be dots and range in number from one to a multitude, they may be fusiform and arranged like the spokes of a more or less complete wheel about the fovea as a hub, or they may be blended into fantastic forms. The picture thus produced is spoken of generally as the **stellate figure** of the macula, and is met with much more commonly in albuminuric than in any other form of retinitis, but sometimes it occurs in other than renal diseases, such as diabetes, tumor of the brain, acute infectious diseases, lead poisoning, and arteriosclerosis. According to *Parsons,* and also *Wilbrand* and *Saenger,* the stellate figure has been observed in cases of retinitis for which no cause could be found. Furthermore, it must be borne in mind that it is not necessary that the white spots should be congregated in the macula, or should form fleecy clouds about the papilla, in an albuminuric retinitis; they may lie anywhere in the fundus, they very often form groups along the courses of the vessels in the vicinity of the papilla and macula, and they may be entirely absent.

Nothing about the ophthalmoscopic picture can give us any idea concerning the variety of the renal lesion, or the stage of the disease. Retinitis occurs most commonly in the chronic interstitial variety of nephritis, but is met with in other forms as well. In the majority of cases it appears late in the course of the disease, when it may be considered as apt to foretell approaching death, but occasionally it is an early symptom and then the prognosis is better. When it appears early the patient may recover from both the renal and the retinal lesions, but blindness may ensue if the latter has been very intense. A young woman suddenly became nearly blind and was found to have a double choked disk caused by an acute nephritis, the presence of which had not been suspected. Some months later, after she had recovered from her renal trouble and apparently was in good health, she was totally blind, and each papilla presented the picture of neuritic atrophy.

Albuminuric Retinitis of Pregnancy

When a pregnant woman slowly loses her vision without any contraction of the fields or loss of the color sense, we are apt to find an albuminuric retinitis. She may exhibit only mild symptoms

of nephritis, and suffer little disturbance of her general health, yet her condition is one which is very serious. If the onset of the retinitis takes place during the **later** months of her pregnancy she may go on to term without any great loss of vision, though it is likely to remain somewhat impaired, or she may have at any time a sudden attack of uræmia in which her vision may be blotted out completely, to be restored more or less only if premature labor is induced immediately. When the onset occurs during the **early** months, and her general condition is so good that an abortion does not seem to be justified, her vision is likely to fail steadily until the pregnancy is ended, either at term or during an attack of uræmia, while a secondary degeneration takes place in the inflamed retina which renders the impairment of her vision irreparable. This form of retinitis ordinarily attacks a primipara, and recurs with each succeeding pregnancy, resulting each time in an increased loss of vision. It is not always possible to tell in these cases whether the trouble is wholly dependent on the changed condition of the organism, or is due to a stirring up into greater activity of a chronic nephritis by the pregnancy, and we shall find nothing in the eye to guide us to a decision.

Diabetic Retinitis

Although a diabetic retinitis may not be distinguishable from any other ophthalmoscopically, it is not hard to diagnose because it is one of the later symptoms of diabetes mellitus, and is met with almost exclusively in bad cases. The great majority of the patients are known to be suffering from this disease before the onset of the ocular trouble, and when this is not the case an odor of acetone about the patient is likely to give us a hint. A point that may help us in a doubtful case is that the hemorrhages generally predominate over the white spots, yet either the hemorrhages or the white spots may be absent. Sometimes we find a central scotoma, because the papillomacular bundle of nerve fibers is particularly apt to be affected in this disease, but whenever there is the least doubt the diagnosis must rest on the finding of sugar in the urine and the presence of other symptoms.

Syphilitic Retinitis

We must take syphilis into account in every case of retinitis in which the ætiology is not clear, for it may manifest itself through a

simple diffuse haziness of a part of the retina, a neuroretinitis with white spots and hemorrhages, an occlusion of the vessels of the retina, or a chorioretinitis. Our suspicions are directed strongly toward this disease when we find a neuroretinitis with a large retinal œdema that extends widely and obscures the papilla, perhaps hiding it from view; when we find the white spots to be more or less pigmented; when some of the arteries are sheathed; and when we are able to detect a diffuse opacity in the vitreous as an accompaniment. The veins may have white margins also, but the walls of the arteries are particularly apt to be thickened so as to conceal the blood columns in places, or to cause a partial or a complete occlusion, but this picture of sclerosis is not diagnostic, it is merely suggestive.

When the fundus of a young person is **pigmented diffusely,** especially in the periphery, so that it looks as though it had been dusted with snuff; or presents a speckling with little black and white, yellowish, or yellowish red spots, as if it had been sprinkled with pepper and salt; or white or yellowish spots with or without pigment in their centers are to be seen, we look at once for other signs of hereditary syphilis, as this is very apt to be the cause, whether we wish to call the condition retinitis or choroiditis. When the onset of the trouble happens about the age of puberty we look to see if the macula presents degenerative changes, as then it may be a case of macular degeneration, or, if both the vision and the intellect began to fail at about the age of six or eight, it may be one of maculocerebral degeneration.

The problem of differentiating a syphilitic retinitis is complicated by the fact that it may appear in a patient who is known to be suffering from some other organic disease, and that such a disease can excite a retinitis of its own in a person who has been infected with syphilis. Unless we are able to discover some fairly characteristic signs that incline us to favor one of the two diseases, or secure improvement through treatment of the one and not of the other, we can do little better than to make a tentative diagnosis from the preponderance of the general symptoms, which may or may not be right.

Saturnine Retinitis

A similar difficulty is present in retinitis due to **chronic lead poisoning,** because albumin commonly is present in the urine of these patients. The diagnosis has to be one of probability rather

than of certainty, but if the occupation of the patient is one in which he is exposed to lead poisoning, if he presents typical toxic symptoms, like wrist drop, a blue line on the gums, and if he has lead in his urine, while his renal symptoms are subordinate, we pronounce the retinitis to be saturnine rather than albuminuric.

Anæmic Retinitis

In very rare cases of anæmia and chlorosis we meet with a retinitis which we are not able to ascribe to any other cause. The whole fundus is paler than common, the papilla lacks its usual luster and has a grayish or yellowish tone. White spots are said to be rare. A neuroretinitis leads us to suspect chlorosis, while numerous hemorrhages are suggestive of a pernicious anæmia, but a similar retinitis with white spots and hemorrhages has been met with in cases of cancerous cachexia. The diagnosis is made through an examination of the blood, and the exclusion of other possible causes.

Leucocythæmic Retinitis

When we see slightly elevated white spots that are edged with red in the periphery of an orange colored fundus, which is splashed with hemorrhages and is supplied with arteries and veins that are of about the same rosy color, leucocythæmia is suggested whether the urine contains albumin or not. Both arteries and veins are dilated, but the veins to a much greater degree. Sometimes the latter are enormously distended and very tortuous.

Gouty Retinitis

There is no ophthalmoscopic picture which is characteristic of gout, but when an elderly patient has white spots and hemorrhages scattered about in his fundus, sclerosis of the retinal vessels, urine that is of a high specific gravity and contains an excess of uric acid, perhaps with albumin, but with no casts, we should inquire at once concerning other symptoms of this disease, which is competent to cause the condition.

Hemorrhagic Retinitis

Hemorrhages form the most prominent symptom in the fundus in two different conditions. One is attended by other signs of in-

flammation of the retina, and we call this hemorrhagic retinitis. In the other there are no other inflammatory signs, and then we speak of **hemorrhages into,** or **apoplexy of the retina.** It is hard to draw a distinct line between these conditions in every case, because the other signs may be quite indistinct, though they are present, and the hemorrhages themselves sometimes seem to excite inflammation. When the extravasations have followed traumatisms to the eye or the head, severe compressions of the thorax or abdomen, or extensive burns, we are safe in pronouncing them simple hemorrhages into the retina, but in most other cases we have to look very sharply if we wish to exclude a retinitis.

The hemorrhagic form is met with in rare cases of almost if not quite every disease capable of exciting a retinitis, but more commonly it occurs when there is serious disease of the heart, or of the blood vessels, and then, as a rule, it is unilateral. *Schmidt-Rimpler* says that he has seen it produced by tumors of the orbit, and it is the most prominent symptom in venous thrombosis. In young people we have to think of nephritis, diabetes, pernicious anæmia, an acute infectious disease like influenza or mumps, chlorosis, and changes in the composition of the blood. It is said to have been caused by obstruction to the venous circulation, by poisoning with phosphorus, and by vicarious menstruation, but it is certainly possible that in some of the cases reported the hemorrhages were without an accompanying retinitis.

THROMBOSIS OF THE CENTRAL VEIN

When one eye suddenly becomes blind, or nearly so, without known cause, and we find in the fundus a large number of dark red, lumpy, and striated hemorrhages, some small, others large, often with little round ones intermixed, all grouped about and over very dark, broad, and tortuous veins, one of which may be seen perhaps to end in a pool of blood, while the arteries are small, and the margins of the papilla are hidden, partly by an œdematous swelling of the retina and partly by the hemorrhages, we diagnose a thrombosis of the central vein of the retina, or of one of its branches. If the hemorrhages are scattered about in all directions the thrombus is in the central artery itself, but if they are confined to a certain area it is the branch that drains this region that is occluded. Grayish patches are to be seen between the hemorrhages.

As time passes the small extravasations become absorbed and white spots appear in the midst of the larger ones, caused by the aggregation of the leucocytes, or sometimes by the formation of crystals. A dark patch of pigment rarely is to be seen. Convolutions of vascular loops are visible on the papilla, perhaps in the surrounding retina, which are anastomoses formed to relieve the engorgement.

This condition may be difficult to differentiate from a hemorrhagic retinitis, except through the extreme engorgement and tortuosity of the veins, which are quite disproportionate to the other signs of inflammation. The only other condition for which it can be mistaken is the possible, though rare, partial occlusion of the central artery attended by great retinal hemorrhages, and this cannot be differentiated with certainty.

Thrombosis of the veins is met with usually in elderly people who are suffering from sclerosis of the retinal and other vessels, heart disease, or some lesion that obstructs the venous circulation, but it may occur in orbital cellulitis, and has been ascribed to the various causes of hemorrhagic retinitis. Once in a while we meet with a case in which the cause defies detection. An example of this was seen in an apparently healthy middle aged lady who had a thrombosis of the central vein of her left eye. A searching investigation failed to reveal any organic disease whatever, and repeated examinations of the fundus failed to disclose any signs of an angiosclerosis of the remaining vessels. She had been overworked and overworried for some time, but such a cause does not seem adequate. The most reasonable explanation seemed to be a strictly localized endophlebitis that had given no previous symptoms—but this is pure hypothesis.

PIGMENTARY DEGENERATION OF THE RETINA

When a person does not see as well as he should in reduced light, as during a cloudy day, or in the evening, the sensitiveness of his retina to light is subnormal and we say that he has **hemeralopia.** Such a condition may be met with in persons who are extremely debilitated by starvation or any exhausting disease, or have suffered from such an infectious disease as malaria, and sometimes it is present in myopes, at least to a minor degree, but when these causes have been excluded we have to search for some pathological condition of the retina that has impaired its sensitiveness.

Concentric contraction of the visual field of a hemeralopic patient, whether the central vision is reduced much or not, suggests pigmentary degeneration of the retina, or retinitis pigmentosa, as it is commonly though erroneously called. This diagnosis is positive if the fundus is found to have a bluish gray tint, and to present in its periphery a network of jagged, **irregular black spots,** many of them shaped like bone corpuscles, some stellate, others looking like granules, which tend to follow the courses of the vessels and to overlay them in places, while the vessels themselves are very small, and the papilla has a yellowish tone and slightly hazy margins. As the disease progresses the pigmentation draws closer and closer to the macula and the papilla, until finally it covers the former and surrounds the latter. At the same time white spots appear among the black, perhaps with little shining dots here and there, the patient becomes nearly or quite blind, and the appearance of the fundus closely resembles that produced by a very extensive old choroiditis, but the differentiation is not so very difficult when we consider the slight details. A creamy yellow, waxy color of the papilla, and retinal vessels which are so small that the veins cannot be distinguished from the arteries and are covered in places by the black spots, establish the diagnosis of pigmentary degeneration of the retina, while a similar yellowish tone of the papilla with vessels that are larger, though smaller than normal, and the passage of the latter across the black spots, are indicative of choroiditis.

So long as the vision remains sufficiently good to enable us to make a perimetric examination of the field the marked concentric contraction is a great aid in the differentiation of this disease. As a rule this contraction is quite considerable before the central vision begins to fail, is much more regular and uniform than in choroiditis, and sometimes is so great that the patient seems to see through a tube.

The picture of pigmentary degeneration of the retina is not always as typical as the above. One variation is the **total absence of pigmentation,** when the patient has all of the other symptoms, hemeralopia, concentric contraction of the field, small vessels, and finally a yellowish papilla, and many intermediate cases between the two extremes of typical and of no pigmentation are to be met with.

Another variation is that a baby may be **born blind** with no visible lesion in his eyes to explain the amaurosis, but within a year

we may see pigmentation develop, the vessels become small, and a yellow optic atrophy supervene.

A few cases have been reported in which the ordinary signs of pigmentary degeneration were complicated by the presence of discrete, round, **white spots** scattered over the fundus, which enlarged and blended while atrophy of the choroid became evident. The white spots may be separated by strips of normal choroid, or nearly the entire fundus may show the picture of choroidal atrophy. This combination of retinal and choroidal atrophy has received the name of **gyrate atrophy of the choroid and retina.** It seems to be a variety of pigmentary degeneration in which the atrophy of the choroid becomes unusually prominent.

In rare cases numerous bright white spots of variable size take the place of the pigmentation in the periphery, and present the same appearance as the **retinitis punctata albescens** of *Mooren,* by which name the picture often is called. Our knowledge concerning this class of cases is quite limited, but this punctate condition of the periphery seems to occur in at least two distinct diseases. In *Mooren's* case there was no hemeralopia, the peripheral vision was normal, and central vision was reduced. Other cases have been accompanied by hemeralopia, concentric contraction of the field, and late failure of central vision. *Roemer* states that he saw typical pigmentation develop in a case of this nature. When we add that the disease seems to be congenital and hereditary, the probability is increased that such cases are atypical forms of pigmentary degeneration of the retina.

The **cause** of this degeneration is not known. The disease is apt to be hereditary, and to occur in several members of the same family, but isolated cases are not uncommon. Usually it is congenital, or develops in early childhood, so that its victims have hemeralopia from the first, but rare cases are on record in which the onset took place in adult life. Consanguinity of the parents and syphilis have been quoted as causes, but at best they are questionable. Other congenital defects often are present. Males seem to be more subject to the degeneration than females. Almost invariably both eyes are affected about equally, though the disease may be confined to one eye in rare cases. The progress is slow, and sometimes seems to be stationary for a long period of time, but no arrest can be ascribed fairly to any form of treatment. The prognosis is bad, and blindness supervenes during middle life, as a rule. A few cases

become complicated with glaucoma, and posterior polar cataract may develop, though this does not happen as frequently as in choroiditis.

AMAUROTIC FAMILY IDIOCY

Occasionally we meet with a Hebrew child not many months old who is becoming increasingly sluggish, and whose muscles are becoming paretic gradually. When we touch him he is apt to struggle so hard that an examination is a matter of difficulty, yet he relapses into a sort of stupor as soon as we cease our efforts. We find his pupils dilated and sluggish in their response to light, perhaps completely irresponsive, and he may have nystagmus. Examination with the ophthalmoscope reveals a cherry red spot at the fovea surrounded by a broad whitish area that does not extend to the papilla, a picture that reminds us of that of an occlusion of the central artery, but this condition is excluded by the practically normal vessels and the absence of any opacity about the papilla. The disk is pale and atrophic. Inquiry is apt to reveal that other children in the same family have suffered in the same way.

The cause of this disease, which is called amaurotic family idiocy, is not known. The affection seems to be confined to the one race, and only a single case is on record in which the child lived to be more than two years old. The only conditions with which it is likely to be confounded are occlusion of the central artery, which is readily excluded by a study of the details of the picture, and maculocerebral degeneration, which occurs in other races and at a later age.

MACULOCEREBRAL AND MACULAR DEGENERATION

When the history of a more or less idiotic child is that he was ordinarily healthy and intelligent until he reached the age of six or eight, the time of his second dentition, when he began to grow stupid and at the same time to lose his power of vision, it is probable that he is suffering from a degeneration of his retinæ and of his brain. This probability is increased if we find that other children in the family are suffering in the same way, though we are likely to search in vain for evidence of heredity. It is not necessary for all of the children in the family to be thus afflicted, on the contrary

the rule seems to be that some are spared, so far as we can judge from the small number of cases that have been reported.

Oatman made a special study of these cases and divided them into two classes, one in which the onset appeared at the time of the second dentition and both the eyes and the brain were affected, and one in which the onset was at the age of puberty and the degeneration was confined to the retina. He called the former **maculocerebral degeneration,** the latter **macular degeneration.** The ophthalmoscopic picture is the same in both. I had the privilege of watching his cases while they were under his observation, and can draw no better sketch than the one he has left us in The Fundus Oculi. The following is his description of the younger of the two children, a boy of eight, in whom a failure of vision and of intellect had been noticed two years before.

"He is in excellent health; no paralysis; no malformation. Vision in right eye 10/200; in left 8/200. Small central scotoma for green and red; peripheral fields normal for white and colors; central fixation; talks well and memory good. Right eye:—Media clear. The macula is encircled by a transversely oval ring of granular pigment, measuring about one disk diameter in length. The enclosed area has a dirty yellowish cast and contains dark spots that can be resolved into fine black granules. The surrounding retina is covered with dustlike pigment. The optic nerve is white on the temporal side. Retinal vessels are narrowed. The left eye presents the same picture as the right, except that the macula within the pigment ring is more atrophic and the fundus as a whole is somewhat lighter in color. Four years later. The disease has steadily advanced. No paralysis. Central scotoma is now absolute. Peripheral field appears normal. Memory poor. Is apathetic and makes little effort to see. Eccentric fixation with eyes upward. Retina is becoming depigmented. Has had five epileptiform convulsions, the first of which occurred three years ago."

The condition in the eyes of the older child was more advanced and optic atrophy was clearly present when she was first seen, at the age of twelve. Her vision and her intellect had begun to fail when she was seven, and she was completely imbecile before she died at about the age of seventeen. These children were brother and sister. An intermediate sister was normal at the age of fourteen. In both of these patients the Wassermann and the von Pirquet tests gave negative results.

This affection of the eyes is always bilateral and may be differentiated from amaurotic family idiocy by the fact that it appears at a much later age. This alone might hardly suffice were it not for the fact that so far as we can judge the pathological changes are not the same in the two diseases. Furthermore, maculocerebral degeneration occurs seldom if ever among Hebrews, who seem to have almost, if not quite, a monopoly of the other disease.

In another family we may find children whose vision begins to fail with the development of the same ophthalmoscopic picture at about the age of puberty, but with no indications of cerebral involvement. This is macular degeneration, which seems to differ from the maculocerebral only in the preservation of the intellect, and the age of onset.

We have to ***differentiate*** these conditions from congenital defects, inflammations of the retina or of the choroid, and other forms of degeneration of the retina. A defect in the macula may be congenital, may be present in several members of the same family, and may or may not be bilateral, but it does not make its appearance after the child has enjoyed good vision, it does not progress or induce optic atrophy, and it is not associated with a progressive loss of the intellect. A central retinitis is excluded by the pigmentation of the retina, while in central choroiditis the black and white spots in the macula are not enclosed in a defined oblong, and are not likely to be associated with a dusky pigmentation of the surrounding retina, such as is common in this disease, or these diseases. When the choroidal markings are visible within the affected area we have to deal probably with a choroiditis, or a sclerosis of the choroid. The history of previously good vision, and the absence of symptoms of hereditary syphilis, suffice to show that the dusky pigmentation of the retina is not the same as that of the snuff colored fundus. Pigmentary degeneration of the retina is excluded by the absence of hemeralopia, the early failure of central with preservation of peripheral vision, instead of the reverse, the early appearance of a central scotoma, and the white rather than yellowish color of the papilla after optic atrophy has set in. We can often trace the hereditary nature of the trouble in pigmentary degeneration, but this has not yet been done in either the maculocerebral or the macular. When idiocy attends pigmentary degeneration it is congenital, but in maculocerebral it seems to develop with equal steps along with the failure of vision. The picture presented in circinate degeneration

is quite different and this condition seldom is met with in childhood.

CIRCINATE DEGENERATION

Occasionally we see in or about the macula of an elderly patient, whose central vision has been rendered imperfect by the presence of a relative, or of an absolute scotoma, an elliptical, usually incomplete zone of irregular round spots, over which the retinal vessels pass. Sometimes these spots are found to have coalesced so as to form an irregular, lobulated design which resembles a yellowish white exudate. Hemorrhages are present, as a rule, but there is no optic neuritis. This condition commonly is called **circinate retinitis,** but it runs a very chronic course and seems to be a degeneration rather than an inflammation. It is seldom seen before the age of fifty, but it has been met with in childhood. This form of degeneration is bilateral in about one half of the cases, and is more common among women than among men.

An atypical retinitis must be excluded by the failure to detect any indications of a disease that can produce such a lesion by a thorough physical examination, which includes not only an examination of the urine, but also the biological laboratory tests, and the observance of clinical signs. Central senile atrophy is marked by white spots that are not grouped in the same manner, and are interspersed with pigment. In a child a possible glioma or tubercle is excluded by the preservation of some vision, and the fact that the surface is elevated very slightly if at all. Colloid formations on the vitreous lamella of the choroid are round, discrete, do not coalesce, and do not impair the vision. Retinitis punctata albescens affects the periphery of the retina rather than the macula, and presents an altogether different picture.

CHOROIDITIS

When we find a sprinkling of black, or of black and white spots in the fundus; a tessellation of a portion of the fundus together with some spots of pigment, some choroidal vessels that have been changed more or less completely into white bands, and a recent loss of vision; yellowish white, or black spots, over which the retinal vessels pass unchanged, except perhaps for a little rise, not accompanied by signs of much retinitis; or one or more white spots that

are edged with or contain black accumulations of pigment, we infer a choroiditis. This diagnosis becomes practically certain if we find at the same time opacities in the vitreous, or perhaps at the posterior pole of the lens. The conditions of coloboma, conus, posterior staphyloma, and rupture of the choroid, which have been described already, may need to be differentiated. A coloboma of the choroid lies in the lower part of the fundus as an oval or parabolic patch that may or may not include the papilla, is congenital, and is differentiated by its appearance, the anomalous courses of the retinal vessels, the depression of its floor, and usually the presence of other malformations, such as coloboma of the iris, and anomalies of the papilla. A coloboma of the macula is distinguished by the history of congenital amblyopia, which has not changed, or the presence of much better vision than could be expected from the appearance of the lesion, the regularity of the outline of the latter, and sometimes the depression of its surface. A white crescent at the temporal margin of the papilla that looks like a widening of the scleral ring, or a narrow white circle about the papilla which is broadest at its temporal side, probably is a conus. A wider crescent or ring in the same situation in a myopic eye, which contains traces within it of the markings of the choroid, probably is a posterior staphyloma. A white crescent at the lower margin of the disk is an inferior conus. A rupture of the choroid is known from its long, narrow, crescentic form, taken together with a history of traumatism. The presence of pigment in and about a white spot usually indicates that the trouble is in the choroid, but there is no rule applicable to all cases by which we can decide with certainty that it is not in the retina.

A lesion in the choroid manifests itself through the changes it produces in the retina. The first effect may be to cause the pigment epithelium to disappear and lay bare the vessels of the choroid, or it may be the formation of an opacity which is followed by a destruction of the pigment epithelium, usually with atrophy of the choroid itself. In some cases much, in others little of the pigment set free accumulates in lumps in and below the retina. If the black spots thus formed are lumpy and are crossed by the retinal vessels they are situated either deeply in the retina, or in the choroid, and we are accustomed to assume them to be in the latter, but if they lie about or cover these vessels we know them to be in the superficial part of the retina. According to *Nettleship* black spots that form a lacelike pattern are always in the latter situation. Quite often

both deep and superficial accumulations of pigment are to be found in the same eye.

Choroiditis is apt to form a part of a **uveitis** in which the symptoms referable to the ciliary body and the iris may or may not be pronounced. Sometimes the symptoms of an iridocyclitis attract the more attention, and the accompanying choroiditis escapes notice, or the lesions in the fundus may be hidden by the opacities that have formed in the vitreous. In other cases the iridocyclitic symptoms are not very marked and may be overlooked if we are not on our guard. It is a good rule to investigate the iris whenever we see a patch of choroiditis, especially when it is situated not far from the papilla. Even when the iris is healthy, the pupil active, and the aqueous clear, we can sometimes find deposits on the lower part of Descemet's membrane that show the ciliary body to be affected. *Griffith* has called attention to such a class of cases in which he has found tuberculosis, or a tuberculous family history, as a rule, but has never found syphilis to be the cause. *Lawson* has described a similar case which he attributed to gout.

A broadcast sprinkling of black dots, perhaps associated with areas of tessellation, or of white dots mingled with black, which mark respectively the **snuff colored** and the **pepper and salt fundus,** is by no means common, but we are much more likely to find circumscribed areas, especially in the lower portion of the periphery, where such a sprinkling is to be seen. This appearance is very suggestive of hereditary syphilis.

In another case we may see one or more yellowish white spots with ill defined margins over which the retinal vessels may rise, but with no other signs of a retinitis to account for their presence, or there may be one or more black spots, each surrounded by a light colored zone; both of these conditions indicate an **early stage** of choroiditis. As time goes on pigment appears in and about the white spots, or white appears in the midst of the black ones, and the change progresses until both have been transformed into pigmented white spots, which give us the picture we see most commonly. These **black and white spots** may be large or small. Sometimes they look as though the tissues had been punched out down to the sclera with an instrument that had left a blotchy black stain at their edges; sometimes they contain patches of pigment, or reveal the markings of the choroid more or less clearly. Occasionally they are pale rather than white, reveal the choroidal vessels dimly, and

contain a dull, grayish black pigmentation, or have similar dull collections of pigment in the retina at a little distance from their edges. In still other cases the choroidal vessels may be plainly visible, and we may have difficulty in distinguishing those areas in which the pigment epithelium has been destroyed from those of normal tessellation, but ordinarily the condition of the choroidal vessels themselves furnish us a ready means of differentiation, as when they are diseased some of them at least are apt to look yellowish or white, and to contain either no blood, or slender red columns, which proves them to be sclerotic or atrophic. When their atrophy is well advanced we may see nothing of them but white bands outlined by the choroidal pigment, and as time goes on this pigment gradually disappears until a clear white surface is left. The center of a very large patch of such choroidal atrophy may be white, while its periphery is yellowish, or of a dirty yellow, and the markings may fade away from the periphery toward the center. These choroiditic spots may be discrete and widely separated, may be arranged in groups, or may be blended so as to cover a large part of the fundus, and it happens very often that spots exhibiting various stages of the disease are to be seen in the same eye. All varieties of choroiditis are likely to induce finally a **choroiditic optic atrophy,** in which the papilla has a yellowish tone, margins that are slightly blurred, and small vessels.

Several varieties of choroiditis are described, some of them named from the sites of the lesions, others from the ætiology, but there is a great lack of uniformity in literature with regard to the way in which some of the names are applied. The term **disseminated** is applied by some writers to all cases in which the lesions are elsewhere than in the macula, while others confine its use to those in which they lie wholly in the equatorial zone. When the foci are confined to the periphery of the choroid *Fuchs* calls the condition an **anterior** choroiditis. *Foerster* used the word **areolar** to designate the cases in which the spots appear first in the region of the posterior pole, where they are for the most part black, and later spread into the equatorial zone, but many call any choroiditis areolar in which the lesions tend to form groups. When the macula is affected primarily or alone, we call the choroiditis **central.** We may say that a choroiditis is **diffuse** when the spots are large, tend to coalesce, and spread over large areas, but many authors fail to see the need of this distinction and use the terms diffuse and disseminated inter-

changeably. In one variety, of which our knowledge is not very extensive, a patch is formed near the margin of the papilla, and is associated with a sectorshaped defect in the field which proves that the lesion has seriously impaired the conducting power of the optic nerve fibers of the retina which pass through it. This is called **retinochoroiditis juxtapapillaris.** *Verhoeff* has reported a case which was caused by syphilis, but we do not know the ætiology in other cases in which this disease was excluded. A choroiditis may be named very properly from its ætiology, after the latter has been ascertained, for this gives the final and complete diagnosis of the case, but such a classification is not convenient for description, because the main purpose of our diagnostic efforts is to determine the ætiology. In tuberculosis alone are the lesions likely to present characteristic features. In order to try to avoid the confusion that has resulted from the introduction and the varying use of so many terms we may perhaps be justified in dividing choroiditis into two main classes, the **simple** and the **purulent,** and then in subdividing the former into only the ***central,*** the ***disseminated,*** and the ***tuberculous.***

Central Choroiditis

When a recent failure of vision is found to be due to the presence of a central scotoma, which is to be accounted for by any of the lesions that have just been described situated in the macula alone, we say that the patient has a central choroiditis. The lesion may be large or small, but is is proportionate to the size of the scotoma. We may see minute black, or black and white dots, perhaps interspersed with brilliant points caused by crystals of cholesterin, or with minute hemorrhages. We may see a number of yellow dots clustered about the fovea, and the nature of these is not to be determined instantly. If a neuroretinitis is present their nature is explained at once, but if there is none we must notice whether we can see any specks of pigment, or if any of the choroidal markings are visible. When neither of these things are to be seen we may question whether the case is one of Tay's choroiditis, or of central retinitis, but colloid deposits on the vitreous lamella of the choroid are excluded by the presence of the scotoma. When pigment spots or exposed choroidal vessels are to be seen the only question is whether we have to deal with senile degeneration of the macula, or with a central choroiditis. In the latter the spot may be quite small,

very large, or of any intermediate size. We may see an area in which the choroidal vessels are laid bare that extends even to the papilla, and the presence within this of any sclerotic vessels suffices for the diagnosis. Occasionally we see a large white spot edged with black, or one or more small ones, when we may need to exclude a possible coloboma of the macula. When a spot is quite small we may be able to perceive radiating out from it a network of fine lines.

The majority of cases of central choroiditis occur in **myopia,** and this condition may be accepted as the cause after we have excluded syphilis, tuberculosis, traumatism and senile degeneration. A dusky, snuff colored, or a pepper and salt pigmentation of the fundus is very suggestive of hereditary syphilis, but the question whether the lesion is due to this disease, or to tuberculosis, must be answered in the way described several times already. Traumatism can be eliminated as a possible cause only by its absence from the history.

Senile Degeneration of the Macula

When an elderly patient presents an area in the macula in which apparently healthy choroidal vessels are visible, separated by dark intervascular spaces, perhaps surrounded more or less completely by a ring of very minute bright points, with or without minute black specks within the area, or shows a number of minute black and white spots about the fovea, while all the rest of the fundus is normal, and we can discover no organic disease to explain such a condition, we say that he has a senile degeneration of the macula. This is included here as a variety of central choroiditis not because it is positively known to be such, but because such accumulations of pigment, as well as the central baring of the choroidal vessels is supposed to indicate a destruction of the pigment epithelium of the retina, and the onset of a senile atrophy of the choroid. The same picture may be presented in the early stages of a central choroiditis that has been caused by myopia, syphilis, tuberculosis, nephritis, or diabetes, so the influence of these diseases must be excluded.

Disseminated Choroiditis

When one or more of the characteristic lesions are to be found in the fundus outside of the region of the macula we say that the patient has disseminated choroiditis, always provided that he does not

present the symptoms of pigmentary degeneration of the retina. The vision of the eye varies with the extent and the situations of the lesions. It may be pretty good, with scotomas in various parts of the field that correspond to the locations of the foci of inflammation, or very bad when the macula has been invaded, or optic atrophy has set in, but we are not likely to find a central scotoma.

Syphilis is responsible for the great majority of the cases, the hereditary form in children, the acquired in adults, but it is not for all. Sometimes the appearance of the lesion gives us a hint as to its cause, but, as in nearly all of the morbid conditions of the fundus, this cannot be depended on, and we are obliged to search out the ætiology through a thorough investigation of the organism. We begin by looking for the stigmata of syphilis, and then make a Wassermann, or a luetin test. A snuff, or a pepper and salt, fundus suggests syphilis so strongly as to be almost diagnostic. If the spots are pale yellowish instead of white, have a dull pigmentation within them or in the surrounding retina at a little distance from their edges, and reveal the choroidal vessels dimly, our first thought is of tuberculosis instead of syphilis, and we resort first to the tuberculin test. Several elevated yellowish white spots, with no marked retinitis and no pigmentation, in the fundus of a very sick person suggest miliary tuberculosis. When the choroidal vessels are plainly visible over a considerable area with little or no abnormal pigmentation, it is possible that the only way in which we can differentiate the condition from a physiological tessellation may be through a history of a recent impairment of vision and the finding of a corresponding defect in the field, but we are likely to find that some, perhaps many, of these vessels have white margins, or have been changed into white bands. Possibly we may have to deal with a senile degeneration in some of these cases, but the cause may be syphilis, or nephritis. *Oatman* has reported a case in which the cause was diabetes. My impression is that some heaps of pigment can be found in all cases of pathological tessellation; they were nearly absent in *Oatman's* case, but two or three lumps can be seen in the stereogram, though they were not mentioned by that author.

The **less common causes** of disseminated choroiditis are numerous and do not seem to be related. *Hutchinson* tells us that it may appear as a **hereditary family disease** independent of syphilis, which emphasizes the importance of not basing a diagnosis on a single symptom. Both traumatism and myopia are competent to

excite changes that produce similar pictures. We meet with a disseminated choroiditis occasionally in women who are pregnant, when it is associated with albuminuria and apparently is an extension to the choroid of the albuminuric retinitis of pregnancy. At different times it has been ascribed to malaria, kidney disease, diabetes, rheumatism, gout, various acute infectious diseases, affections of the nose and pharynx, malnutrition, anæmia, and chlorosis. *Ball* attributes it in young, healthy subjects to excessive near work of the eyes, whether the refractive errors have been corrected or not. We are pretty sure that it can be caused by intestinal disorders, though we cannot always obtain such positive evidence as *Kirkendall* did when he saw an obstinate case of disseminated choroiditis get well after an operation for appendicitis; we certainly do see moderate improvement occasionally follow treatment directed to the intestinal tract. We can always determine with fair certainty whether the patient is suffering from syphilis, tuberculosis, or any other demonstrable organic disease, but sometimes we are not able to discover any such disease, and then the ætiology of the choroiditis is apt to remain unknown.

Tuberculous Choroiditis

Some writers make a distinction between a diffuse inflammation of the choroid, which they hold to be referable to a toxæmia and call tuberculous choroiditis, and a circumscribed metastatic lesion caused by colonies of bacilli, which they term tuberculosis of the choroid, but although this differentiation may become valuable in the future, at present it has little bearing on prognosis or treatment, and is to be made clinically with difficulty, or not at all, in the majority of cases.

When we examine the fundus of a very sick patient, usually a child, whose symptoms are indicative of acute miliary tuberculosis, or of meningitis, and find a number of round or oval, yellowish white, elevated spots about half as large as the papilla, which have ill defined margins, we believe that the spots mark the presence of **miliary tubercles in the choroid.** Occasionally we may see at the same time a brilliant white elevation of a part of the papilla with a grayish œdema of the adjacent retina, which we take to be a tubercle, or a conglomerate of tubercles, of the optic nerve. It seldom happens that we are able to follow the development of these

lesions long, as the patient is apt to die within a few days, but we meet with similar spots in the chronic forms of tuberculosis, and may see them either disappear under treatment, or change so as to present the picture of disseminated choroiditis. In these cases the origin of the lesions may be detected by the tuberculin test.

In another child we find a yellow, gray, green, or blue **tumor,** as large or larger than the papilla, which projects into the vitreous. If such a tumor is without vessels and reacts distinctly to the tuberculin test, glioma of the retina and sarcoma of the choroid are excluded, and we know that we have to deal with a solitary, or a conglomerate tubercle of the choroid. One that develops from a single focus is called ***solitary,*** one that is formed from the combination of several foci is ***conglomerate;*** when the tumor is very large it is spoken of as a **tuberculoma.** It is possible that we may have the opportunity to view the fundus of a child for two or three successive days, and to see a little, yellowish white spot increase in size so rapidly that within a couple of days it has formed a tumor which has a diameter nearly equal to that of the papilla, and very likely changing its color in the meantime; or to observe two or more little foci blend, change color and form a similar tumor. The rapidity of its growth marks such a tumor as tuberculous.

PURULENT CHOROIDITIS

When a patient who is suffering from septicæmia that is derived from any source, such as puerperal fever, ulcerative endocarditis, caries of the bones, or from any of the acute infectious diseases, like measles, scarlet fever, mumps, tonsillitis, cerebrospinal meningitis, influenza, diphtheria, pneumonia, or typhoid fever, has a rapid and total loss of vision, either with or without a severe attack of iridocyclitis, we have good reason to fear that septic emboli have lodged in the choroid. If the case is seen very quickly we may find the appearance of the fundus to resemble that observed in septic retinitis, or some yellowish spots may be visible, but within a few hours everything in the fundus has become hidden by opacities in the vitreous. Panophthalmitis is apt to develop rapidly, but sometimes, oftenest in children who are suffering from meningitis, the angry symptoms of a generalized suppuration do not develop, a yellow mass becomes visible in the vitreous, where it remains quiescent, and then becomes organized and vascular. Finally the newly

formed tissue shrinks irregularly so as to form a brassy yellow mass, that may need to be differentiated later from a glioma, and is called a pseudoglioma.

GLIOMA OF THE RETINA

A mother brings a child to us because she has noticed something yellow in one of his pupils. The child is in ordinarily good health, has no pain or redness of the eye, the anterior media are clear, and the tension is normal. The pupil may be of normal size, or slightly dilated, but as it does not respond to light we know that the eye is blind. The yellow reflex from the pupil and the blindness gave this condition its old name of **amaurotic cat's eye.** With the ophthalmoscope we find a yellowish or reddish yellow, elevated, lobulated mass in the vitreous, perhaps spotted in places with lustrous white spots, covered with ramifying blood vessels, some of which may be embedded in the tissue, and sometimes the whole surrounded to a greater or less degree by a bluish or greenish detachment of the retina. The very important question must be decided at once whether this is or is not a case of glioma.

This tumor of the retina is not common, and is not easy to differentiate, yet its early diagnosis is extremely important, as it is very malignant and the only chance the child has for life lies in enucleation during its early stage. At the same time the enucleation of the eye of a young child should be avoided whenever possible without endangering his life, or running the risk of sympathetic ophthalmia. Glioma occurs almost wholly in infants, or in very young children, though it has been met with as late as the eleventh year. Several members of the same family have been known to be affected, but no hereditary predisposition to its occurrence has been demonstrated. It may appear first in one eye and then in the other, or it may be confined to one alone. The average length of life after its discovery is about a year and a half, and the only recoveries on record have followed early enucleation.

At a **very early stage** the tumor may be small enough to allow us to see considerable of the fundus, and then we may find it surrounded by a grayish ring that looks like œdema, little hemorrhages in the retina or about the papilla, and black and white spots that are indicative of choroiditis. We seldom see this picture, for in the great majority of cases the glioma is further advanced when it is brought to our notice, but when we do we must make sure that the

mass is ***distinctly elevated*** above the surface of the surrounding retina, because there is at least one case on record in which an eye enucleated for glioma proved to have nothing more serious the matter than a circinate retinitis. In this latter disease the macula may be the seat of a yellowish white exudate, but the vision is not abolished completely, and the pupil reacts to light. As it might be possible for the pupil to react to light when the glioma is so very small, it is wise to keep a child with a yellowish mass in the fundus under observation long enough to enable us to decide whether it is increasing in size or not. If it increases with extreme rapidity and responds to the tuberculin test it is a tubercle, if it grows less rapidly and does not respond to tuberculin there is little doubt that it is a glioma.

In **most cases** the vitreous is occupied by a smooth or irregular yellowish mass, which may be bluish or greenish in places, or with lustrous white spots, and may or may not be provided with vessels. The clinical picture may be modified by ill health of the child, possibly but not necessarily connected with the trouble in the eye; by pain, which we refer to the eye if symptoms of glaucoma are present but not otherwise; by an irritation of the eye, or perhaps by posterior synechiæ and other signs of a present or past iritis, minus tension, or secondary glaucoma.

The lustrous white spots mentioned as seen sometimes on the surface of the yellow mass mark where bits of the tumor have crumbled off, and if this disintegration has progressed sufficiently we may find the vitreous to be filled with crumbled yellowish fragments.

When the mass is very far forward, and yet the anterior chamber, pupil and tension are normal, a congenital posterior polar cataract with a persistent hyaloid artery and a vascularization of the posterior capsule of the lens must be excluded, for an eye with such a condition has been enucleated for supposed glioma. First we ***test the reaction of the pupil,*** for if the pupil reacts to light glioma is excluded; if it does not react we know that the eye is blind, but must dilate the pupil and study the lens, for if the clear portion of this seems to be abnormally thin, or if we can make out streaks of opacity in its posterior part, we have to deal with a congenital defect.

When the mass is behind the lens a bullous **detachment** of the retina is a possibility. This is very rare in children, and can be differentiated ordinarily by its bluish or greenish color, yet *de*

Schweinitz and *Shumway* have reported a case of total detachment in a boy two years old that presented a yellowish pink mass in the vitreous over which some large vessels could be seen. It could not be differentiated from glioma, the eye was enucleated, and the retina was found to contain changes which were ascribed to a dropsical degeneration of the nuclei and protoplasm of the rod and cone visual cells. We cannot expect to be able to differentiate such an exceedingly rare condition.

The bluish white **cyst** caused by the tænia or the cysticercus can scarcely be mistaken for a glioma, as it is neither yellow, lobulated, nor vascular, yet it needs to be mentioned as one of the conditions from which a glioma must be distinguished.

A **sarcoma** of the choroid resembles a detachment of the retina more than it does a glioma, yet a whitish, lobulated tumor over which the vessels of the retina pass may be a leucosarcoma, and this may give rise to a whitish reflex which resembles that of a glioma somewhat, although its color is not typical. Our only means of identification in such a case may be through a close examination to determine whether any of the vessels are embedded in the tissue or not, but no great harm is done if we fail to make the differentiation, as both of these tumors are very malignant and demand the same treatment.

If the eye presents the symptoms of iridocyclitis, if it has been wounded, or if the patient is suffering from septic disease elsewhere in the body, the yellow mass may be an **abscess of the vitreous** following a purulent choroiditis, or caused by direct inoculation with septic material. This diagnosis is positive if we find pus in the anterior chamber, or if panophthalmitis develops rapidly.

A solitary or conglomerate **tubercle** of the choroid sometimes forms a yellowish mass that extends into the vitreous and resembles glioma closely. It is not vascular, while glioma commonly is, and in rare cases we are able to recognize it through an extremely rapid growth, but, as a rule, a response to the tuberculin test is the only positive means of differentiation, though we always take into account the presence elsewhere of symptoms of tuberculosis. After glaucoma has set in, or after the vitreous has become filled with granulations, we may be absolutely unable to distinguish between the two. In such a case we can only consider that a tubercle is less common than glioma, and that a clinical differentiation is not essen-

tial to treatment, as the eye should be enucleated no matter which tumor is present.

The most difficult question we have to answer is whether we have to deal with a glioma, or with an inflammatory exudate called a **pseudoglioma.** We must gather every particle of evidence that may point in either direction, and consider it all carefully before we decide. We inquire whether the child has suffered at any time from meningitis, from one of the acute infectious diseases, from a traumatism to the eye, and whether the eye has ever been noticed to be red and inflamed. If the child has ever had any of these things he may have had an attack of uveitis that destroyed his sight and left a yellowish exudate in the vitreous. Signs of iritis enhance the probability of a pseudoglioma, but we must not forget that a child may have any of these troubles, even a severe uveitis, without the formation of such an exudate, and subsequently develop a glioma. We feel confident of pseudoglioma when the tension is minus, but when the tension is normal or plus we must study the color, the surface, and the vascularity of the mass itself in search of distinctive characteristics.

The color is not decisive, but an exudate is likely to have a brassy yellow hue, that is, a metallic tone, while a glioma is apt to be of a softer reddish yellow, and to show patches of white, with perhaps a greenish tint. Both may be associated with a large bluish or greenish discoloration that indicates the presence of a detachment of the retina. The surface of an exudate usually is smooth or ragged, that of a glioma lobulated, but this rule does not always hold true, for the shrinkage of an exudate may take place in such a way as to cut it up into lobes, while particles of a glioma are rather apt to break off in such a way as to leave a jagged surface. Most exudates are not vascular, while most gliomas have vessels coursing over them, but again this is not true of every case. When some of the vessels lie almost hidden because they are embedded in the tissue, the diagnosis of glioma may be said to be almost certain, but embedded vessels are not always to be seen. The symptoms in typical cases may be summarized in the following manner:—If a child who has a blind eye that emits a yellow reflex from the pupil has a history of disease and presents symptoms suggestive of a possible former attack of uveitis, and we find the tension of the eye to be subnormal, while we are able to see in the vitreous a brassy yellow, smooth or ragged mass that contains no vessels, we diagnose pseudoglioma.

If on the contrary there is no history of disease or injury, there are no symptoms referable to a former uveitis, while the tension is normal or plus, and the mass in the vitreous is reddish yellow, perhaps spotted more or less, lobulated, and vascular, with some of the vessels evidently embedded in the tissue, the diagnosis of glioma is fairly positive. Unfortunately variations in these symptoms appear in nearly every case to cloud the differentiation, so that the most expert diagnosticians often are in error.

When the tumor has reached a more **advanced stage** we shall find an emaciated child who is evidently very sick and is suffering intense pain. The eye is likely to be enlarged, deformed, and protruding, with an enlarged cornea that is bordered by a bluish zone, or by an anterior staphyloma of the sclera, and sometimes with episcleral nodules along the vortex veins, or the anterior ciliary vessels. The cause of these symptoms of infantile glaucoma is understood if we find a glioma in the other eye, or the cornea remains sufficiently clear to enable us to see into this eye and recognize a tumor. In the latter case we may not be able to determine the nature of the tumor if the other eye is not affected. In another child this stage may have passed and the pain have diminished suddenly with the appearance either of an exophthalmos, or of a fungus-like growth on the anterior surface of the eyeball. We may find the orbit distended, a swelling of the preauricular gland, or one that involves the region of the parotid and extends down over the angle of the jaw, or little metastatic tumors may be present in the forehead, or the scalp. Sometimes convulsions or paralyses lead us to conclude that metastases have taken place into the brain. In any such case it is evident that the eye is the seat of a malignant growth and that the hope of saving life is extremely small, if not nonexistent, whether the tumor be a glioma, a sarcoma, or a tuberculoma. In a little child the chances are rather in favor of its being a glioma, but the differentiation had best be left to the pathologist.

DETACHMENT OF THE RETINA

When a patient tells us that he has scintillations, sparks, flashes, or balls of fire before his eyes, we know that his retina is irritated. When he says that objects appear to be bent, broken, or jagged, we suppose that the rods and cones are slightly displaced. Such symptoms lead us to think of a commencing detachment of the

retina as a possible cause, the probability of which is increased if hemeralopia has developed recently, and if blue objects appear to be green. But such **prodromal symptoms** often escape notice, and the first intimation the patient receives that anything is wrong may be the sudden appearance of a **dark cloud** before his eye. This cloud may obscure a portion of whatever object he happens to look at, and sometimes is observed to wave with the movements of the head, or it may render the eye either practically or totally blind. Such a history as this, taken together with a defect in the field that corresponds to the cloud when the vision is sufficiently good to enable us to detect it, a deep anterior chamber, and minus tension, leads us to expect to find a bullous detachment of the retina. In another case the history may be one of a gradual **impairment of vision,** perhaps with the development of a similar cloud that may or may not have changed its position

When the media are clear we perceive readily with the ophthalmoscope a gross change in the fundus, but there is no lesion of the eye in which a systematic use of this instrument is more imperative if we are to get anything like an accurate idea of the actual condition present. The important details cannot be brought out at any one step in the examination, for we are obliged to see a part at a time and to group these parts together mentally so as to get a composite picture of the whole. This is why we never see anything exactly like the illustrations in the textbooks, which necessarily are composite and represent the conditions as they are, rather than as they appear in either the upright or the inverted image at a given focus.

After the pupil has been dilated we look for the **red reflex** and observe it while the patient turns his eyes in various directions. This reflex should be bright and clear from all parts of the fundus, so if we see dark spots float across it, which we recognize as caused by opacities in the vitreous, and its color changed to a bluish or a greenish gray in one or more places, we know that we have to deal with either a detachment of the retina, a tumor of the choroid, or a dense exudate into the vitreous. If the red reflex is blotted out, or is replaced by a grayish one, we have to determine whether this is due to a large detachment, a large tumor, or a hemorrhage into the vitreous. A hemorrhage usually can be differentiated by the history and by a dark red reflex obtained by oblique illumination. Frequently this is all that we can learn in this way, but sometimes

we are able to get an approximate idea of the size and site of the discoloration, and occasionally we can see in the latter crooked black lines, as well as whitish bands that alternate with darker ones and change slightly so as to give the impression of a wavy movement. When we see this picture we know that the waviness must be caused by a fluctuating bullous detachment.

Next we introduce a convex lens a few inches in front of the eye and use the **indirect method** of examination, by which we can definitely exclude an exudate by the absence of its characteristics and the presence of dark retinal vessels running over the surface of the discoloration. An exudate is excluded likewise if there is no history and no sign of a previous inflammation of the eye that might have been a uveitis. We can also determine at this time the size of the discolored area, its relations to the papilla, whether its margins flatten out into normal retina or seem to overhang the latter, and whether its elevation is slight or great, sufficiently at least to inform us whether it is flat or bullous. When it is bullous we may be able to see the surface wave, or fluctuate when the patient moves his eye, which assures us of the presence of serous fluid. Frequently an aperture can be seen through which the markings of the choroid are visible, usually in the upper peripheral part of the discoloration, which we recognize to be a **laceration** of the retina. Instead of such a picture as this we may see a wavy, fluctuating gray mass with crooked black lines running over it, or perhaps we see the papilla at the bottom of a gray funnel, particularly in a case in which we were unable to obtain a red reflex, and then we know that we have to deal with a **total bullous** detachment of the retina. If there is no such mass visible, but the entire fundus is of a bluish gray with white bands here and there over which the dark retinal vessels bend, the case is one of a **total flat** detachment. The papilla may be perfectly normal, but if the detachment approaches it closely its margins are likely to be obscured so as to cause us to think of an optic neuritis, but this lesion is excluded by the size of the retinal vessels, which is about normal, though they are dark, tortuous, and without light streaks. A similar condition that affects only a part of the retina is a **partial flat** detachment. An œdema of the retina which is sufficiently pronounced to be taken into consideration is of a grayish white rather than a bluish or greenish gray, is associated with some such lesion as an optic neuritis, a choked disk, or an occlusion of a retinal vessel that decides its nature, and the vessels behave differ-

ently. A carcinoma of the choroid may induce a detachment that is rather flat, but this is attended by hemorrhages in the retina, and is almost invariably secondary to carcinoma elsewhere in the body.

By the **direct method** we then measure the height of the elevation, and trace the courses of the vessels as they pass over it, by interposing successively stronger plus lenses in the ophthalmoscope, while at the same time we observe its individual features. If the detachment is flat we go farther in determining whether it has or has not been caused by a carcinoma by observing the presence or absence of a yellowish color and of spots of pigment. In rare cases we may in this manner first perceive a detachment of the retina by a clear serous fluid that produces no discoloration, by observing that the retinal vessels bend forward, lose their light streaks, and mount upward in a tortuous manner into the vitreous, where they seem to be without any support.

When the discolored area has a high elevation the most important question for us to answer is whether it has been caused by a **subretinal effusion,** or by a **tumor of the choroid.** If it is known to have started above and to have gravitated downward, the retina reattaching above as it became detached below, and particularly if it has left a laceration above through which the markings of the choroid can be seen, it is a simple detachment, but in many cases we do not have the advantage of such a history, and we may not be able to find a laceration. Folds in and fluctuation of the surface indicate the presence of serous fluid, but do not exclude the possibility of tumor, while their absence, leaving a smooth, nodular swelling, leads us to suspect tumor strongly, especially when new vessels or hemorrhages are present, or the tension of the eyeball is increased, but this is not a positive indication. The most reliable means of differentiation now at our disposal is a test of the translucency of the swelling. We may place an electric light in the mouth of the patient and observe whether the swelling appears to be light or dark in comparison with the rest of the fundus; if it is light we assume that the detachment is due to the presence of serous fluid, but if it is dark we know that either a tumor or a hemorrhage lies beneath the retina. A better way is to use a Sachs transilluminator. We place its tip against the sclera beneath the swelling and observe whether the pupil becomes illuminated or not when the light is turned on; if it does we conclude that the detachment has been caused by serous fluid, if it does not that the light has been intercepted by

either a tumor or blood. We may infer that a hemorrhage has taken place if the eye has been subjected to traumatism recently, if there is evidence of vascular disease, or if glaucoma existed in the eye previously, otherwise we feel confident of the presence of a tumor. The differentiation between a simple detachment of the retina and a tumor of the choroid may be made perfectly in the majority of the cases in which the media are clear, yet it is hard to speak with absolute certainty. Some years ago I enucleated a hard, painful, blind eye that contained a smooth, nodular, bluish protrusion into the vitreous, which showed no signs of fluctuation on its surface, and was dark on transillumination. The diagnosis of tumor of the choroid, probably sarcoma, seemed to be positive, but the pathologist reported it to be a simple detachment.

Occasionally we meet with a case in which there is no discolored area in the fundus, and the only objective evidence of the trouble is an **abrupt arching forward** of the retinal vessels into the vitreous, where, dark and without light streaks, they seem to hang, branch, and curve without support. This is a simple detachment in which the retina and the serous fluid are so transparent as to transmit the red reflex from the fundus beneath them. It has been referred to already as the form in which we may first recognize the nature of the lesion when we examine the eye by the direct method.

Detachment of the retina occurs rarely in children, and when it does its diagnosis is usually attended with no more difficulty than in adults, but a couple of possible peculiarities may be mentioned. As a rule, a detachment lasts a considerable time before it becomes reattached spontaneously, but a few cases have come to my knowledge of puzzling transient detachments in children who were suffering from kidney disease, and in at least one of them a very serious renal lesion was not discovered until after several of these had occurred, so the suggestion may be in place that the kidneys of any child should be investigated in whom a detachment occurs spontaneously, especially if it is transitory. The other pecularity is one that was met with by *de Schweinitz* and *Shumway* in a child in whom the retina was so yellow from degeneration that a glioma was counterfeited clinically, and the correct diagnosis could not be made.

Thus far we have been considering cases in which the refractive media were clear, but the diagnosis of a detachment of the retina, and still more its differentiation from a tumor of the choroid, becomes very difficult whenever the lens, or any other of the refractive

media, is too opaque to allow us to see into the eye. Sometimes we can infer the presence of a detachment from a faulty projection associated with a minus tension, but we have to exclude all other diseases that may destroy a part of the field of vision. Detachment is excluded if the field is contracted concentrically. A nasal contraction with normal or plus tension directs our suspicions in the direction of glaucoma, though it is possible for a detachment or a tumor to occupy the temporal portion of the retina. If the field can be taken accurately enough to show that a half or a quadrant of the field is wiped out in both eyes, with the blind part delimited by a fairly straight line, we look for a cranial lesion that has caused hemianopsia. It is only when the defect in the field seems to be rounded or irregular, light is projected in erroneous directions, and the tension is minus, that we feel pretty sure of the presence of a detachment. When the tension is plus we think of a tumor of the choroid, and sometimes Sachs's transilluminator is of great assistance when it is used in a dark room, provided that the opacity of the media is not too dense to permit the passage of some light from within outward. We move the tip about from place to place on the sclera, and if we find that the illumination of the pupil is cut off when the tip is at a certain point we feel fairly sure that a tumor is present. The diagnosis and the differentiation must be made in nearly every case through the weighing of every particle of evidence that can be obtained.

DETACHMENT OF THE CHOROID

A few days after a cataract extraction we frequently find the eye quite soft, the anterior chamber shallow or empty, although the wound has not reopened, and we can see, either by oblique illumination or with the ophthalmoscope, a yellowish or brownish mass behind the iris that has no folds and does not fluctuate. Sometimes several such masses are to be seen. We need not be worried about such a case, the choroid has become detached, but the prognosis is good and we can confidently expect it to return to its normal position in a few days.

After a contusion of the eye we can sometimes see by the curved courses of the vessels that a part of the retina has been elevated, although it retains its normal appearance in general, and functionates perfectly perhaps, because its relations with the choroid are

maintained. In such a case we may suppose that the choroid has been detached from the sclera by a hemorrhage, though the latter is not visible unless the fundus is albinotic.

Detachment of the choroid may be caused also by a great loss of vitreous, or by the contraction of cicatricial tissue in a blind eye which has been the seat of a very severe intraocular inflammation, but on the whole, with the exception of the last mentioned class of cases, it is of much less serious importance than detachment of the retina, and is apt to escape observation. It is usually recognized through a protrusion of normal retina into the vitreous in an eye which is soft and has a shallow anterior chamber.

TUMORS OF THE CHOROID

Every distinct elevation of the surface of the retina leads us to think of a possible tumor. If the patient is a young child, the surface of the swelling is smooth or lobulated, and its color is yellowish, the tumor may be a glioma of the retina, or a tuberculous growth of the choroid, both of which have been discussed already, while if the color is bluish or greenish we have to determine whether it is a detachment of the retina, or some tumor, probably a **sarcoma,** of the choroid. Whether the patient is a child or an adult the cases are few in which we can definitely determine the nature of a tumor of the choroid, or even whether it is benign or malignant, prior to the enucleation of the eyeball, but while it is possible for us to meet with a gumma, an angioma, or possibly some other neoplasm, such benign tumors are extremely rare, while sarcoma is common, and the differentiation very seldom can be made clinically. We may suspect a growth in the choroid to be a **gumma** when the patient has tertiary syphilis, and prove it to be such by watching it melt away under appropriate treatment, or the presence of other vascular anomalies might go far to convince us that a certain protrusion was caused by a congenital **angioma,** but in the great majority of cases the demonstration of a tumor of the choroid is equivalent to a diagnosis of sarcoma.

A tumor of the choroid needs to be differentiated from a **cyst** of the retina, or of the vitreous, on rare occasions, when we see a bluish white growth with semitranslucent walls, especially when it is somewhat obscured by fine, membranous opacities in the vitreous. We suspect such a growth to be a cyst if it has a generally translucent

appearance, and this diagnosis is positive if it has a slightly tremulous movement, or if we can see a small white spot within it.

When the history given by the patient is that of a slow, painless impairment of vision, that may or may not have culminated suddenly in the blindness of one eye, and we find a more or less globular, bluish or greenish protrusion of the retina into the vitreous from any part of the fundus, we suspect that it is caused by the presence of a sarcoma in the choroid. If it has a light pink, or flesh color we may believe it to be a leucosarcoma, but, as a rule, the various histological varieties cannot be differentiated ophthalmoscopically. A sharply defined scotoma, and a tense, smooth surface of the swelling, taken together with the history, may make us practically certain of our diagnosis, but very often enough detachment of the retina is added to make us uncertain, and sometimes the differentiation of a small tumor in the midst of a large detachment is quite difficult. A soft eyeball inclines us to believe that no tumor is present, while a hard one tends to strengthen our suspicions, but the tension is at best an unreliable guide. The best means of differentiation that we possess is transillumination, which has been described already under detachment of the retina.

Once in a while we meet with a **carcinoma** of the choroid, and it is important that we should be able to differentiate this from sarcoma. If the patient has had a rapid loss of vision, so that one or both eyes have become practically blind within two or three weeks, especially if there has been much pain in the eye that was not accounted for by any externally visible conditions or by an increase of the intraocular tension, and we see near the posterior pole a yellowish elevation that slopes away gradually into the surrounding tissues, probably associated with a more or less extensive detachment of the retina, which may be flat, or if a detachment is the only thing visible with such a history, we suspect a metastatic carcinoma of the choroid. This diagnosis may be considered fairly well established if we can find a primary cancer somewhere else in the body, for in this situation it is almost if not quite invariably secondary. The primary growth usually is to be found in the breast, but it may be in some other part. The patients almost always are elderly.

A **flat sarcoma,** or an **endothelioma,** may resemble a carcinoma quite closely, particularly when it is not pigmented and is situated near the posterior pole of the eye, but the history is that of a slow

loss of vision, and we are unable to find a primary growth in any other part of the body. The differentiation between the two is important, because the indications for treatment are exactly opposite. Unless necessary to relieve pain enucleation is contraindicated in carcinoma of the choroid, because the operation seems to shorten life rather than to prolong it, while it is indicated in sarcoma as the only means by which life can be prolonged.

CHAPTER XVIII

Amblyopia, Affections of the Color Sense, and Defects of the Visual Field

The literal meaning of the word **amblyopia** is subnormal vision without any apparent fault in the eye, but the term commonly is applied also to many other conditions in which the sight is impaired either by congenital faults, or by lesions which are now known, but were not formerly recognized to be characteristic of disease, or of poisoning, even when gross changes are present in the fundus. The vision may be normal for black and white and yet subnormal for colors, so amblyopia may be said to range in degree from a slight reduction of the vision below the accepted standard, or a slight defect of the color sense, to total blindness. **Amaurosis** means literally total blindness without obvious cause, so it may be understood to be the extreme limit of amblyopia, though the word often is used synonymously with blindness from any cause. Amblyopia may be either congenital or acquired; in the latter case it is due in most cases to disease, traumatism, or poisoning, and if we look carefully we shall find in many some indications of trouble in the fundus, or possibly that the amblyopia is confined to a central area of the field, when the patient has what will be described presently as a central scotoma. Whenever objective signs are absent, so that the eye looks perfectly well, we must exclude malingering, especially when the patient is an adult, as we do not always know in what way benefit may accrue from a diagnosis of amblyopia to a person who claims to have faulty vision in one or both eyes, but we must never pronounce him to be a malingerer until we have proved him to be such.

Ordinarily when we speak of amblyopia or amaurosis we refer to a lack of perception throughout the entire field of vision, but in many other cases we find areas of total, or of partial blindness in various parts of the field, which may or may not be perceptible to the patient, but can be detected, measured, and delimited by means of the perimeter. These are spoken of generally as **defects** in the

field of vision. When the blindness is total in the affected area the defect is said to be ***absolute,*** when the light sense is preserved it is called ***relative.*** A relative defect may be for color alone. An island of absolute or relative blindness in the field is called a **scotoma.** A triangular area with the papilla at its apex is a **sectorlike defect.** Absolute or relative blindness of one half of the field is **hemianopsia.**

AMBLYOPIA

CONGENITAL AMBLYOPIA

We seldom if ever find the visual power of the two eyes to be exactly the same in any person after all of the refractive errors have been corrected, and it is well known that the visual acuteness varies considerably in different people, but we consider such variations to be physiological and in no way indicative of amblyopia as long as the vision is equal to or better than the standard which has been accepted commonly as normal. It is only when the vision of an eye has been found to be subnormal after all of the refractive errors have been corrected, and we are convinced that the condition has existed since early childhood, that we make a diagnosis of congenital amblyopia.

In many cases only one eye is affected, and this is, or has been strabismic; then we may debate whether the amblyopia was congenital, or has resulted from disuse, without much hope of arriving at a conclusion. Sometimes the amblyopia may have been caused by injury to the retina by hemorrhages at the time of birth, but such cases seldom can be differentiated. Defects of structure account for some, and when these are bilateral the patient is apt to have nystagmus. A considerable degree of hypermetropia, or of compound hypermetropic astigmatism, may render one or both eyes amblyopic. When this trouble is bilateral we can often obtain a great improvement in a few months through the constant wearing of glasses that correct the refractive errors, but though we may occasionally secure a similar improvement when only one eye is affected, through making it work after its refractive errors have been corrected, we are not often able to do so, and when we cannot it is hard to determine whether the amblyopia is due to this, or to some other congenital cause. If the patient never had strabismus, has no great

refractive error, and particularly if he has binocular single vision, we must exclude all forms of acquired amblyopia, as well as malingering, before we can accept it to be congenital.

ACQUIRED AMBLYOPIA

When the sight of an eye is known to have been good, and then to have been impaired or lost, either suddenly or gradually, we are able in most cases to find some lesion in it to which we can attribute the loss, but occasionally we cannot, and then we say that the patient has acquired an amblyopia, the cause of which we then try to ascertain. Sometimes we can detect some slight changes in the fundus that do not seem to us to be competent to explain the deterioration of the vision, but the only cases in which the lesions in the fundus are gross and prominent to which this term is applied are those that have had the symptoms produced by the toxic action of drugs, and these are all included under the name toxic amblyopia. Acquired amblyopia is met with occasionally in only one eye, but as a rule it is bilateral.

Traumatic Amblyopia

When a patient complains of amblyopia or amaurosis as the result of a traumatism to the head or spine, but presents no lesion in the fundus or elsewhere in the eye, we must search not only for symptoms of an intracranial lesion that might affect the centers of the optic nerve, but also for signs of any general disease which might produce a similar condition, look for stigmata of hysteria, and exclude malingering. The last is met with most often in connection with court cases in which the plaintiff desires to secure unduly high damages, but is to be excluded painstakingly in all cases, as we cannot tell what harm may result from a failure to unmask an impostor. When all of these things have been excluded we are justified in pronouncing the condition of the patient to be a traumatic neurosis.

Amblyopia from Bright Light

When a person has been exposed for a considerable time to the reflection of the sun's rays from the surface of snow or water, or has been close to an electric flash caused by a short circuit, or has

been exposed to such a bright light as that produced in electric welding, he may have a distinct impairment of his vision either with or without any discoverable lesion in the fundus. As a rule the amblyopia is temporary, but in bad cases it may be permanent, or blindness may be produced. Gazing at the sun, usually at the time of an eclipse, likewise is able to induce an amblyopia, which sometimes is attended by a central retinitis. Ordinarily we find a relative central scotoma for color in these cases, though sometimes the scotoma is absolute, and we may find the color vision defective elsewhere in the field, while some patients complain of metamorphopsia. *Swanzy* says that when the cases are not severe improvement takes place in the vision, but complete recovery is not common, and that no case in which the vision was reduced below 6/18 has been known to regain 6/6.

Amblyopia from Disease

In the absence of any history of poisoning, traumatism, or exposure to a bright light an acquired amblyopia is an indication that should lead us to a thorough investigation of the organism for all diseases that may induce trouble in the retina, or in the optic nerve. A careful inspection of the fundus may reveal very slight changes that escaped our notice at first, such as a slight haze that directs attention toward syphilis or nephritis, a hyperæmia that suggests malaria, or an uncertain pallor of the temporal sector of the papilla. If we think that possibly we have seen the last we test the central vision for a scotoma for red, which may be the first symptom of a diabetes, or a symptom of multiple sclerosis, but always suggests much more strongly poisoning with alcohol and tobacco. If the patient is very ill he may be suffering from ptomaine poisoning, also called botulism, or he may be in a state of exhaustion from some grave disease, either of which is a sufficient explanation, or he may have had a large spontaneous hemorrhage, which for some unknown reason is more apt to induce amblyopia than an equal loss of blood from a wound.

Reflex Amblyopia

Cases have been reported in which the cause of an amblyopia has been proved to be reflex by its disappearance as soon as an apparently unrelated source of irritation was removed. Carious teeth were at fault in the majority of the cases, but troubles in the nose,

intestines, genitourinary organs, and perhaps elsewhere, have been quoted as the causes. The diagnosis can be made only after relief has been obtained, but the possibility of a reflex cause should be borne in mind whenever we have to deal with a case of acquired amblyopia that seems to be inexplicable, and a search should be made for a source of irritation.

Hysterical Amblyopia

When a patient complains of blindness of one eye, while the media and fundus are perfectly normal, we suspect hysteria at once. Less often the complaint is of imperfect vision of one eye, but it is only in rare cases that both eyes are affected. The patient usually is a neurotic woman, but may be either a man or a woman of good physique whose appearance does not cause us to think of hysteria. In every case we have to rely upon the finding of concomitant, inexplicable nervous symptoms for our diagnosis. We must be watchful to see that the symptoms we elicit are not those of some organic lesion of the central nervous system, as it is quite possible for an amblyopia associated with anomalous nervous symptoms to be the first sign of such a lesion, and it is only when the symptoms found are irreconcilable with any organic trouble that a diagnosis of hysterical amblyopia is justified.

If a ptosis, or a blepharospasm, appeared coincidently with the amblyopia; if both eyes are in a state of conjugate deviation; if the pupil is dilated, whether it responds to light or not; if the patient suffers at the same time from asthenopia, or from diplopia; or if objects are said to appear to be larger or smaller than they should, we first search for a cause to explain these accompanying symptoms, and if none can be found we feel confident that the trouble is hysterical.

When sufficient vision is present to enable us to use the perimeter we have no doubt as to the diagnosis if we find an irregular concentric contraction of the field for white, which changes rapidly in shape and extent, as shown by the results of several examinations made in succession, and particularly if we find that the fields for color exhibit gross anomalies, such as an overlapping of their margins, or a reversal. The field for red has been found to be larger than that for white in some of these cases and a relative scotoma is common.

When the eye is amaurotic we test the other senses, and if we are

able to find anomalous areas of anæsthesia to touch, pain, or temperature somewhere on the body, a loss of certain reflexes, or anomalies of taste, smell, or hearing, that cannot be referred to a lesion in any part of the central nervous system, we are justified in a diagnosis of hysterical amaurosis. There seems to be a central disconnection between the conduction apparatus of the eye and the percipient function of the brain in these cases, for sometimes we can prove that the amaurotic eye does see by placing a five or six degree prism in front of it, when the patient sees two lights as though the sight in both eyes was normal, yet the patient is not a malingerer, the loss of vision is just as real to her as though the eye was really impaired.

Toxic Amblyopia

A bilateral acquired amblyopia leads us to question the patient quite closely with regard to his **occupation** and **habits,** as well as concerning any recent or long past overdose with any **drug,** in order to try to obtain a clue to the cause. The occupation may lead us to suspect chronic poisoning with lead, bisulphide of carbon, or aniline oil, the habits may reveal a chronic poisoning with alcohol and tobacco, while many drugs are able to impair the vision when an overdose has been ingested. Lesions in the fundus are produced in the great majority of cases, though they are not always typical. **Lead** induces a great variety, that range from no visible changes to a high degree of neuroretinitis with hemorrhages, and we make the diagnosis in such a case from the occupation and the presence of other symptoms of lead poisoning. If we find a pallor of the temporal side of the papilla, with a contraction of the periphery of the field and a central scotoma for red in a patient whose occupation exposes him to the danger of poisoning with **bisulphide of carbon,** we ascribe his toxic amblyopia to this cause. The occupation together with a violet color of the fundus and dark vessels leads us to ascribe an amblyopia to poisoning with **aniline oil.** Disease of the papillomacular bundle of nerve fibers means chronic poisoning with **alcohol and tobacco** in most cases, but it may be caused by poisoning with lead, bisulphide of carbon, iodoform, or cannabis indica, as well as by certain general diseases. A retinal ischæmia with very small vessels suggests that a toxic dose of **quinine** has been taken, while a retinal hyperæmia may indicate a toxic dose of phosphorus, scammony, colocynth, or pomegranate.

Toxic Amblyopia from Quinine

A sudden attack of blindness, deafness, and tinnitus aurium, with both retinæ of a grayish or yellowish white color, and both disks very pale with extremely small blood vessels, forms such a characteristic clinical picture that we have good reason to fear when we see it that the patient has taken an overdose of quinine. In the majority of cases we have no difficulty in making the diagnosis, as we are informed at once that the patient has taken an enormous dose of the drug. Patients differ greatly in their susceptibility to quinine. Some can take doses that may be called truly heroic without apparent harm, while others exhibit toxic symptoms after the ingestion of as small an amount as two grains, and a temporary blindness has been produced by fifteen grains given in divided doses in the course of twenty-four hours. When sufficient vision is preserved to enable us to map out the field we find it much contracted.

The prognosis is commonly thought to be good, but this is questionable. As a rule the vision returns after the drug has been eliminated, the blood vessels refill, and the fundus regains its normal color, but the field is apt to remain contracted. Exceptional cases have been reported in which the patients remained blind for life, but this is unusual. In spite of this return of good vision it has been observed repeatedly that the vision is apt to deteriorate slowly, and that at the end of many years the papilla may be found to be pale, the blood vessels small, and the vision to be quite poor. From the researches of *Holden,* and of *de Schweinitz,* it would appear as though the effect of the drug is exerted primarily upon the ganglion cells of the retina, and that it also produces inflammatory effects in the walls of the blood vessels. These effects may perhaps be sufficient to explain such a slowly progressive atrophy without recourse to the idea of a persistent toxic action of the drug upon the nerve fibers, but we cannot say that the prognosis is good when such an impairment of the eye is at all likely to be the final result.

If the patient denies having taken quinine he may have taken instead a toxic dose of salicylic acid, oil of wintergreen, ergot, some of the coal tar products, filix mas, potassium chlorate, cocaine, adrenaline, nitrobenzene, or dinitrobenzene, all of which are said to be able to induce similar pictures, though they vary somewhat in their details, and usually do not have features that are quite so pronounced.

Toxic Amblyopia from Alcohol and Tobacco

When a middle-aged or elderly patient complains of a gradual deterioration of vision for both distance and near, which cannot be corrected by glasses, and we find a central scotoma for red due to a disease of the papillomacular bundle of optic nerve fibers, the probability, if the patient is a man, is that he has been using both alcohol and tobacco to excess for many years. Usually this fact is admitted at once, and then, after we have excluded the other possible causes of disease of this bundle, the diagnosis is made. Occasionally a patient is untruthful about his habits in that he will deny drinking even when he admits the habitual abuse of tobacco, and, unless we are able to detect the untruthfulness, we may be led to ascribe his condition to tobacco alone. A chemical examination of the contents of his stomach may reveal an alcoholic gastritis and make the diagnosis plain. This form of toxic amblyopia is met with sometimes, but much less often, among drinkers who do not use tobacco, and very rarely among men who have indulged in tobacco for years without drinking. The possibility that tobacco alone can excite disease of the papillomacular bundle has been doubted by a good many competent observers, but it has been proved by a small number of cases in which the diagnosis was indubitable.

It seems to be probable that the same disease can be excited by the excessive drinking of **tea,** possibly of **coffee,** so this is a habit that needs to be inquired into at times, particularly when the patient is a woman.

Wood Alcohol Poisoning

This form of toxic blindness is so unique in its clinical picture that it can scarcely be mistaken for anything else. The history tells us that after drinking some alcoholic preparation, perhaps a household remedy like Jamaica ginger, the patient was seized with vomiting, headache, dizziness, and dimness of vision, or that he had a seizure like this after he had been working for some hours in the concentrated fumes of some such preparation as varnish, very likely after he had first fallen in a stupor. It tells us further that he became nearly blind in about twenty-four hours, that then his vision began to improve, but after a few days started to deteriorate again until it left him totally blind. We find a white optic atrophy. In such a case the diagnosis of wood or methyl alcohol poisoning is positive.

If we happen to see the patient very soon after the ingestion of the poison we find his pupils dilated and irresponsive to light, perhaps with no observable change in the fundus, more probably with an œdema of the retina that may overhang the margin of the disk, or with a neuroretinitis, but within twenty-four hours we are pretty sure to be able to see signs of optic atrophy. If he is not totally blind at this time it is possible that we can make out a central scotoma with a contraction of the fields. A few cases of recovery are on record, but the improvement in vision that takes place within the first few days almost invariably proves transient and illusory, as the toxic action results in optic atrophy, which may be either simple or neuritic.

Amblyopia from Overwork

A diagnosis of this kind is extremely questionable and should not be made until after every other possible cause has been excluded, even when there is no question that the eyes have been abused. We may be able to dispose easily of the questions as to traumatism, exposure to a bright light, disease, and drugs, but we can hardly exclude congenital amblyopia unless there is a record of a previous examination to which reference may be made, and it is almost equally hard to exclude reflex irritation and hysteria. Yet these requirements seem to have been met in a case I reported in the Annals of Ophthalmology for April, 1896.

A young man, twenty years old, consulted me in September, 1892, to see whether his eyes were all right before beginning his studies in the Massachusetts Institute of Technology. Both eyes were in excellent refractive and muscular condition, and the vision of each was 20/15. During the following January, after he had spent the night from 8 p.m. to 7 a.m. in hard work at fine mechanical drawing, he was seized with a pain in his right eye that radiated backward and downward into his neck, and persisted for several months. Three months later, in April, 1893, the vision of his right eye was 20/50, that of his left 20/15 as before. The tension was normal, the fields perfect, the pupils were of equal size and not dilated, and nothing could be found either in the fundus or elsewhere to account for the pain or the loss of the vision. The patient was examined at this time by a number of ophthalmologists of the first rank, who agreed unanimously that they would have pronounced the case to be one of congenital amblyopia of one

eye without a question, if it had not been for the record in my possession. Examination by a neurologist revealed no stigmata of hysteria, and no source of reflex irritation was located, but it was appreciated that neither of these conditions could be excluded with certainty at once. Two years later the vision of the right eye was 20/40 +, in 1915 it was 20/30. No signs of hysteria or of reflex irritation have appeared in these twenty-two years, and every other cause than overwork now seems to have been positively excluded.

MALINGERING

Any person who feigns or exaggerates an illness or an inability in order to avoid work, or to secure some advantage to himself is called a **malingerer,** but in this place we refer only to a simulated defect in the vision of one or both eyes. Quite a number of tests for this kind of malingering have been devised, and a few will be given by which simulation can be detected in most cases. A malingerer always pits his wits against those of the examining physician, and when he is bright he is apt to be well posted in the tests to which he is likely to be subjected, so modifications in the tests often have to be devised on the spot, the object of which always must be to prove whether the defect in vision is real or counterfeit. The position of the physician must be that of an unbiassed judge, and he must never allow his judgment to be influenced by any prepossessions for or against the truth of the patient's claim. The tests vary as the person complains of blindness, or of some degree of amblyopia either in one, or in both eyes.

If the patient claims to be **totally blind in both eyes,** that is, without perception of light in either, we test the reflexes of the pupils to light, for if these are absent we know that the eyes are blind. If the pupils react to light there is a rather remote possibility that he may be telling the truth, so we observe his movements while he walks about the room with his eyes open, then bandage his eyes and have him walk about again; if he is really blind his movements will be the same, but if he is not they will be very different.

If **one eye is said to be blind** we resort first to the pupillary reactions again, for if the pupil of that eye does not react directly to light but does consensually, the blindness is real, while if the pupil reacts directly to light we are almost certain that the eye

sees. Then we have the patient look at a light placed a few feet away and interpose a prism with its base in or out before the supposedly blind eye; if it sees, and binocular single vision is present, it will move toward the apex of the prism, to return as soon as the prism is removed, while it will not move at all if the eye is blind, or lacks ability to fuse its vision with that of the other. Frequently we can detect a malingerer by covering his good eye, and then having him lift one of his fingers and look at it; he will direct the eye straight at the finger if it is blind, because he knows the direction of his own finger, so if the eye looks in any other direction we know that he is malingering. If we bring an object close to his eyes and have him look at it the two eyes will converge, but one is apt to deviate outward in a short time, either because it is blind, or because of a muscular insufficiency; then if we slip a screen before the good eye and the bad one turns back toward the object, we know that it sees. Another test is to place a prism with its base up or down before the bad eye and have the patient walk down stairs with both eyes open; he will have no difficulty in doing this if the eye is blind, but he will if he is simulating.

Instead of blindness the claim may be that the **vision of one eye is subnormal.** It is possible for a real amblyopia to be exaggerated, but when simulation has been detected we seldom interest ourselves in this possibility, the measurement of the actual degree would be a matter of considerable difficulty, and practically we assume that the whole defect has been simulated. After the usual test of the vision of each eye separately we may leave the amblyopic one uncovered and busy ourselves with a pretended examination of the good one, first interposing plain glass and weak convex lenses, then gradually increasing the strength of the latter until the vision becomes blurred, or we feel confident that the limit of his hypermetropia has been passed. In the latter case we cover his supposedly bad eye, change the test card, and direct him to read again; if he cannot do this we know that he was reading with the bad eye, and that his amblyopia is simulated. *Jackson* places two cylindrical glasses of the same strength, one convex, the other concave, with their axes coinciding so that they neutralize each other, before the good eye, allows the patient to assure himself that he can read through the glass, and then pretends to test the bad one with plain glasses; while doing this he manages to displace one of the cylindrical glasses so that its axis crosses that of the other at

an angle and renders vision with the good eye impossible; if the patient does not notice the change he is malingering.

A simulation or an exaggeration of **amblyopia in both eyes** to an equal degree in each is very difficult to detect when the patient is quick witted and well informed. We may succeed in confusing him by means of a variety of test cards on which the characters are presented in so many different ways that he has nothing but their actual size to guide him, and so get statements that are widely at variance with one another. Slight discrepancies may be accounted for by the fact that all letters of the same size are not equally perceptible, but when the same letter or character of a small size is read at one time, but not at another, the variance can be explained only by simulation or exaggeration. Sometimes we can detect simulation positively after a test at twenty feet by having the patient hold a chart with reversed type in his hands, stand ten feet from a mirror, and read the reflected characters; if it is impressed on his mind that this test is at one half the distance of the preceding one he is apt to read much smaller letters than he did before, if he is malingering.

Sometimes a malingerer simulates a **defect in his field of vision,** as a rule a hemianopsia, or a concentric contraction, but such a simulation is not apt to be hard to detect. In **hemianopsia** the eye is blind in one half of the field, though the vision may be good in the other; in marked concentric contraction the entire periphery of the field is blind, so that the patient seems to see through a tube. A rapidly approaching object causes a reflex closure of the lids of a seeing eye, which is difficult to control by the will even when the test is expected, while it produces no effect on a blind one. If we bring an object smartly and unexpectedly toward the eye in the blind part of the field and the patient winks, the defect is simulated. Another test for **concentric contraction** is to measure the limits of the field at a certain distance, and then at a greater one. The measurement in degrees should be the same in both cases, although the actual size of the field is larger at the greater distance, but the malingerer is apt to have the size remain the same, thus causing the field to decrease in degrees with the distance. Such a phenomenon is observed occasionally in hysteria and in the traumatic neuroses, so these have to be excluded before we can pronounce the patient to be a malingerer.

AFFECTIONS OF THE COLOR SENSE

We meet with some curious anomalies when we come to deal with the color sense. Some patients tell us that they see colors where we cannot, others that they are unable to see certain colors. In both cases the anomaly may be a symptom of disease, or of poisoning, or may occur as the result of some unknown cause in persons who otherwise appear to be perfectly well in all of their organs. The subjective sensation of color where the latter does not exist is called **chromatopsia,** or colored vision, while a complete or partial inability to distinguish colors is known as **color blindness.** If we wish to gain even an approximate understanding of the latter we must be familiar with the little that is known about color vision.

CHROMATOPSIA

When a patient tells us that he sees red, blue, green, or yellow colors where they do not exist, our first inquiry is whether this happens only after he has been gazing fixedly at a colored surface, or when his eyes are closed, as in the first case the colors seen are complementary and physiological, and in the other we know that some people are able to see colors when their eyes are shut, probably because of psychical reasons. Except in so far as the latter may be a symptom of hysteria, such phenomena are of no pathological importance, provided that the eyes present no objective lesions, and the vision otherwise is normal. Beautiful colors sometimes float before a blind eye, perhaps because of an irritation of the visual centers, and are apt to excite in the mind of the patient an illusive hope that the vision of the eye can be restored. When after-images have been excluded, the eyes possess good vision, and the colors are seen when they are open, we must learn whether the colors take the form of **rainbow tinted halos** about lights, for then they constitute a symptom of glaucoma. If all objects appear to be tinted we notice whether he is an overworked, nervous, underfed individual, as such a general condition may account for the phenomenon, and whether he shows signs of hysteria, but in every case we must learn whether his eyes have been exposed to a bright light, or his cornea has been wounded, and search his fundus for evidence of disease. Chromatopsia is not uncommon after successful cataract extractions. The

patients may tell us that they see all manner of beautiful colors, or that everything appears to them to be red. Red vision is called **erythropsia.** *Swanzy* states that two thirds of all recorded cases of erythropsia have followed successful operations for cataract, while the remainder of the patients have possessed normal eyes. In the former it usually appears weeks or months after the operation, while in the others it is apt to come and go at irregular intervals. It is usually transient, and the cause is not known, as a rule. Once in a great while we may find a slight intraocular hemorrhage in the form of a film of blood. Attacks of chromatopsia have been attributed to overindulgence in coffee, tobacco, and alcohol, as well as to overdoses of digitalis, cannabis indica, and amyl nitrite, when possibly it may be ascribed to a toxic derangement of the color perceiving center in the brain. If objects appear to be yellow we think first of an overdose of santonin, but the same phenomenon may be produced by chromic acid, picric acid, and digitalis, as well as by such diseases as glaucoma, and optic atrophy.

COLOR VISION

Very extensive and elaborate studies have been made, and are still being conducted concerning this subject, but we cannot claim to know much about it as yet. Quite a number of theories have been advanced to explain the way in which we are able to perceive color, notably by *Hering, Young,* and *Helmholtz,* but none of them can be called quite unexceptionable. The latest, and seemingly the best, is the one suggested by *Edridge-Green,* which, stated very briefly and imperfectly, is that the varying impulses communicated to the sensory nerve endings in the retina are sent to a color perceiving center in the brain where they are analyzed and sorted. If this center is fully developed all of the colors of the spectrum will be perceived by the individual, if it is imperfect only some of the colors will be recognized, and if it barely exists no color at all can be appreciated. Our actual knowledge consists of a few facts from which we draw inferences. Apart from light there is no such thing as color. Light consists of innumerable rays of various wave lengths, a comparatively few excite sensations of color when they fall upon the human retina, while the great majority do not. Most men and women are able to distinguish six colors in the spectrum, or in the rainbow, red, orange, yellow, green, blue, and violet, while a com-

paratively few can distinctly see a seventh color, indigo, between the blue and the violet. No one has yet been known to be able to recognize an eighth color, but if any individual were to claim that he could do so we might be obliged to admit such a thing to be possible, particularly if he had excellent vision of the other seven, because it has been demonstrated that persons whose color vision is supposed to be normal differ enormously in their powers of color perception. We have no more right to assume the extreme limit of the color perceiving center to be fully known, than the most of us have to deny the existence of those who can see indigo as a separate color because to us it is simply a deep shade of blue. Tests calculated to reveal slight differences show that the gradations in the perception of colors cover a very large range, and that distance, as well as atmospheric conditions, affects in different ways the visibility of each. Occasionally a person will confound blue and green at first glance, but distinguish them after a moment. Any one will have difficulty in distinguishing a blue from a violet, or a yellow from an orange light, when it is seen from a distance, or in a fog, even though a red can be distinguished easily from a green one under the same circumstances. Then there is a minority of people who can perceive only five, four, three, or two colors of the spectrum, and a few who are totally color blind. For an exhaustive inquiry into this interesting and complex subject reference must be made to writings that deal with it alone.

Color Blindness

We say that a person is color blind when he is unable to perceive certain colors. If we use the term strictly in this sense it may perhaps be held to apply to nearly every human being, so ordinarily we use it to indicate a defect of the color sense that is sufficiently marked to unfit the person for certain callings. The most common form with which we have to deal is that in which **red** cannot be distinguished from **green,** and it is at the same time one of the most important, because it unfits the individual for service on railroads, or at sea, where red and green lights are used as signals. Equally important, though much rarer, is **total** color blindness, in which no color at all can be perceived, but the whole world is supposed to appear in varying shades of gray. Red is used as a signal light because the red rays are able to penetrate a fog to a greater distance than any of the others, green because it is complementary to

red. If a man is blind to red it makes little difference from a practical standpoint whether he can perceive green or not, that is whether he is a protanope or a deuteranope, he must be placed in the dangerous class so far as signals are concerned. Less important are the rare cases in which blue and yellow look alike, and those in which orange cannot be distinguished from yellow, or blue from green, because such defects are not likely to become sources of danger, although they render the persons incapable of following certain callings in which good color vision is required.

Color blindness may be either **congenital** or acquired. A child who is congenitally unable to distinguish red from green is apt to become aware of his defect very early, and to conceal it with great ingenuity. A gentleman who was thus afflicted said that when he was a boy he was obliged to take lessons in art, and to conceal his defect he labelled his paints, laboriously studied the proportions in which they should be mixed to produce certain shades of green and red, ascertained what colors were needed, and followed directions so carefully that his teacher never expressed any suspicion that green and red looked alike to him. He tried to explain how be was able to recognize colors under ordinary circumstances, which seemed to be largely through an elimination of possibilities or probabilities, but had learned that his method was unreliable through the purchase of a gaudy red necktie under the impression that it was a sober green. These patients study every detail of appearance that can guide them in detecting the presence of a color to which they are blind, and sometimes they become remarkably expert, but it is as hard for them to tell how they do it as it is for anyone to convey an impression of any other sensation. It may be that their ability to sort yarns, to read party-colored letters, and to recognize colored lights, depends upon the nature of the training to which they have subjected their eyes, at least to some extent. Some can pass either one, or both of the first two tests, or pass one to fail in the other, but probably few succeed in passing the lantern test with its mutations of brightness and distinctness, and the recognition of color developed in this way is not reliable. In other cases the defect is not so marked, and the patient may be wholly unaware of its presence until he enters some business in which good color vision is necessary, when he learns of its existence through his mistakes. Such patients are apt to be worried and annoyed by the discovery, and the demonstration of the character of the defect seems to be easy, as a rule.

Acquired color blindness is apt to appear first as a minute central scotoma, to which the patient unconsciously adapts himself by utilizing his parafoveal retina for the recognition of color. As such a scotoma is due almost invariably to disease of the retina, or of the papillomacular bundle of optic nerve fibers, it tends to become larger and to produce a serious impairment of the central vision. Therefore it is very important that we should be able to detect it, not only because it is a symptom of a disease which may not yet have manifested itself in any other way, but also because it is making the patient dangerously color blind. It would be of interest to know what proportion of the persons who pass the lantern test and afterward are discovered to be color blind belong to this class, for, as long as the scotoma is small and the color sense remains unimpaired in the retina about the fovea, it is readily conceivable how the patient might succeed in passing all of the ordinary tests with perfect honesty on his part, and how the defect would become apparent both to himself and to others as the scotoma grew larger. In all cases in which acquired color blindness is demonstrated or suspected the fundus should be examined carefully, for it is not unlikely that we shall be able to find there signs that help to guide us to a general disease, or to a toxic condition of the organism.

Detection of Color Blindness

It has long been claimed that in testing the color vision the patient should not be required to name the colors, because theoretically he might be able to distinguish them clearly without knowing their names, but *Marshall* makes the point that color ignorance does not exist apart from color blindness, that a person of average intelligence who calls primary colors by the wrong names does not see the difference between them, and goes so far as to say that a test cannot be reliable unless the colors are named. It may be well not to insist on the names when we are dealing with a foreigner who is not thoroughly conversant with our language, and possibly with a young child, but aside from these exceptions, *Marshall's* point seems to be well taken. Another point in favor of the naming of the colors is that this is absolutely necessary for the detection of **amnesic color blindness,** in which colors can be seen, recognized, and matched, but cannot be named. Amnesic color blindness is almost if not invariably associated with a right homonymous hemianopsia and other

symptoms of intracranial trouble, which lead us to think that the lesion is probably in such a place in the occipital lobe as to interrupt the tracts connecting the visual and the speech centers.

The commonest test is to match **skeins of colored yarns** in good daylight. A sample skein is handed the patient and he is told to select the different shades of the same color from a considerable number of skeins of various other colors. This test is known as *Holmgren's,* and has been modified in various ways by *Oliver, Thomson, Edridge-Green,* and others. It enables us to detect defects well enough for the ordinary purposes of life, so it is very useful in general clinical work, but it is insufficient when the question of competence to recognize railway signals is raised, because many persons can pass it who are absolutely red blind. In most cases we eliminate the probability of color blindness if the patient matches the colors quickly and correctly, and know him to be deficient if he hesitates and makes errors, but if he is slow in his choice, even though he is correct, he may have to regard certain colors for a moment to identify them, or he may be habitually slow in all of his movements.

Edridge-Green devised another simple test in which the patient is given a tray containing a large number of **beads** of various sizes and colors, along with a pair of forceps and four boxes labeled red, yellow, blue, and green, each with a hole large enough to admit a bead, and is instructed to pick out the beads of these colors and drop each into its proper box. When he has finished the boxes are opened and the result is apparent.

Stilling invented another test in which letters are formed by spots of colors so that a person who is color blind will read them incorrectly, but this seems to be not much more efficient than matching yarns.

The best test yet devised to detect color blindness, which does not consist of a very small color scotoma, is a **lantern** in which colored lenses are placed before a light seen through apertures of varying sizes, and with the additional interposition of smoked, ground, ribbed and otherwise modifying glasses, so as to reproduce as nearly as possible the various conditions in which signal lights are seen in actual service on the railroad and at sea. Quite a number of designs of these lanterns have been invented. A person fails to pass this test if he calls a color by the wrong name, and if he does not see a red, green, or white light that is visible to people with normal sight. Even this has not proved to be perfect, as individuals have

been known to pass it who were discovered, either then or later, to have faulty color vision.

To detect a **central color scotoma** we have the patient fix his eye on a small spot or object which is made to change its color; if he does not recognize the color when he is looking directly at the spot, but does when it is moved a little to one side, or he moves his eye a trifle, he has a scotoma for that color. The test object must be at least equal in size to the minimum required for perception by normal eyes, which varies for different colors and proportionately to the distance. Commonly we use on the perimeter a disk about a tenth of an inch in diameter, which is close to the minimum for violet, and ten times the minimum for red. We can measure the size of the scotoma, if it is large enough, by moving the test object in various directions until the color is perceived and marking the boundaries thus obtained on a chart.

DEFECTS IN THE FIELD OF VISION

THE NORMAL FIELD OF VISION

The field of vision is the extent of space in which objects can be distinguished by an eye while it remains fixed on one point. Its peripheral limits for light and white are greatest, over 90°, to the temporal side, as rays of light which come from that direction in the plane of the pupil strike the cornea and are refracted into the eye, but they are circumscribed elsewhere by the tissues that surround the organ.

Tests of the Visual Field

A rough test of the limits of the field of vision may be made by having the surgeon and the patient sit or stand face to face a short distance apart with their eyes about on a level. To test the patient's right field his left eye and the surgeon's right are covered, and, while each looks steadily into the exposed eye of the other, the surgeon brings his hand, or better a white object on a black rod, slowly in, on a level with the eyes, midway between him and the patient. Assuming that the surgeon's field is normal, the object approaching from the temporal side should be perceived at the same moment by both him and the patient, but if the latter is unable to see it until it has been brought nearer to the median line, his field is contracted in

that direction. The limit to the nasal side, above, and below is learned in the same manner, but it is not apt to agree exactly with that of the examiner, because of differences in the prominence of the features of the faces. This test is made easily and quickly, and by it we can detect any considerable contraction of the field, but it is not able to furnish us much of the information that we need when an abnormality is present. Accuracy in measurement, and in the location of a defect in the field is secured by means of a **perimeter,** which consists of a metal arc marked off in degrees, which rotates about its central point, where it is attached to an upright bar secured firmly to a base or standard. Attached to the same standard with the upright is a head rest which can be so adjusted as to place the patient's eye in the center of the curvature of the arc, his other eye being covered. The central point of the rotation of the arc is marked by an aperture, or a white spot, which serves as a point of fixation for the eye. While the patient's eye remains fixed on this central point a white test object, placed in a carrier, is moved from without inward along the inner surface of the arc until it is perceived, when the degree on the arc at which it stopped is noted and marked upon a chart. The arc is then rotated a certain number of degrees, fifteen, forty-five, or ninety, as desired, and the observation is repeated. After the limit in each meridian has been marked on the chart we connect the points by a curved line, which marks the boundaries of the field of vision for white. The boundaries of the color fields are determined in the same way, after we have substituted red, green, blue, or other colored test objects for the white ones. Scotomata are detected by moving the test object along the arc from the periphery of the field toward the center, when the patient may notice that it disappears at a certain point to reappear at another. By changing the inclination of the arc a few degrees, repeating the test, and noting on the chart the point at which the test object disappears and reappears in each position of the arc, the shape, size, and location of a scotoma can be mapped out accurately.

Measurements of the Field of Vision

As the form and size of the field are influenced by the sunken condition or the prominence of the eye, by the lids, by the upper and lower margins of the orbit, by the shape and size of the nose, and to a slight extent by the size of the pupil, the condition of accommoda-

tion, and the length of the eyeball itself, it is not surprising that the average figures given in various text books do not agree very closely. We may accept those given by *Berry* to be probably as nearly correct as any. These are:—

"Upward, 45°; upward and outward, 50° to 55°; outward, 90° (often slightly more); outward and downward, 80° to 85°; downward, 70°; downward and inward, 60° (variable on account of the nose); inward, 55° to 60°; inward and upward, 55°. The extent of the field upward and upward and inward is found to be 5° to 15° greater when the point of fixation is situated 20° or 30° from the center of the perimeter in the opposite direction." Charts abound in the market in which an approximately normal field has been marked off, but it is evident that the boundaries in any individual case are not likely to agree exactly with those indicated, except that outward the bounding line should always be at 90° or more. The limits of the color fields, likewise are subject to variation, but approximate fairly well with these figures, which have been taken from *de Schweinitz:*—

	Blue	Red	Green
Outward	80°	65°	50°
Outward and upward	60°	45°	40°
Upward	40°	33°	27°
Upward and inward	45°	30°	25°
Inward	45°	30°	25°
Inward and downward	50°	35°	27°
Downward	58°	45°	30°
Downward and outward	75°	55°	45°

We can easily construct a chart for our guidance by tracing the outlines of the various fields according to the above figures on any of those to be obtained in the market, and this will be more useful if the tracing is made with inks that correspond to the colors indicated, using black to delimit the field for white.

CONTRACTION OF THE FIELD OF VISION

When the limit of the field is materially less in any direction than is indicated on such a chart, and this variation is not to be explained by some unusual prominence of the tissues about the eye, the field is said to be contracted. If this contraction affects only one part of

the periphery we look for some local affection of the retina, such as a detachment, unless it happens to be of the nasal portion, when our first thought is of glaucoma. When the boundaries are constricted in all directions we have **concentric contraction,** which is met with in all of the affections that impair the general efficiency of the optic nerve and retina, is well marked in optic atrophy, and may be said to be the commonest functional disturbance of the eye. Its degree is found to vary from one that is not noticed by the patient and can be detected only by perimetric measurements, to one in which the patient is deprived of peripheral vision and seems to see through a tube. Such an extreme concentric contraction as the last with more or less good central vision is quite suggestive of pigmentary degeneration of the retina, but it is met with also in traumatic neuroses and in hysteria. A distinctive feature in these cases is that when such a tubelike contraction of the field is produced by retinal disease the orientation is impaired so badly that the patients are scarcely able to go about alone, while there seems to be little if any such difficulty when the contraction is caused by hysteria or the neuroses. A defect of this nature has been counterfeited sometimes, so a test for the detection of simulation has been given under malingering. A transient concentric contraction has been found sometimes after an attack of epilepsy.

The **fields for color** may be contracted in a similar manner, or they may be distorted, in that they are made to overlap or interlace, or they may be reversed. Distortions and reversals of these fields we usually find to be symptomatic of lesions in the central nervous system, of neuroses, or of hysteria.

A curious phenomenon, which is met with in some cases of traumatic neurosis and might be mistaken for a contraction, is *Foerster's* **displacement of the field.** The distinctive feature of this is that the boundaries of the field are not the same when they are tested by the appearance, and by the disappearance of an object on the perimeter. For example, if we bring the test object into view from the temporal side the patient perceives it at 90°, and after we have carried it past the center he loses it say at 30° on the nasal side; then when we reverse the procedure and bring the object into view from the nasal side he perceives it at 60°, and loses it again at 60° on the temporal side. In such a case we could readily make a mistake and think the field contracted concentrically, if we tested it by moving the test object from the center outward, although no contraction really was

present. It is always best to determine the boundaries of the field by moving the test object from without inward.

SECTORSHAPED DEFECTS IN THE FIELD OF VISION

Sectorshaped defects may be caused by lesions in the retina, or by affections of the central nervous system. When they cut the fields for white and for colors evenly they are apt to be caused by an occlusion of certain of the retinal vessels, or by the interruption of the conductivity of a group of nerve fibers in the retina. A triangular defect with its apex at the papilla, at a point corresponding to the position of a circumscribed patch of retinochoroiditis close to its margin, may be held to be the diagnostic feature which marks the case as one of retinochoroiditis juxtapapillaris, and not one of an accidental location of a patch of choroiditis at this point. Other sectorlike defects are met with in tabes, and in multiple sclerosis, but in these the boundaries for white and for colors are not apt to coincide. Still other defects of this shape may be partial hemianopsias.

SCOTOMATA

An island of absolute or of relative blindness in the visual field is called a scotoma. If it includes the point of fixation it is **central;** if it lies near, but does not include the point of fixation, it is **paracentral;** if it lies at a considerable distance away from this point it is **peripheral.** Sometimes the island forms a more or less complete circle about an unaffected point of fixation, and then we call it an **annular** or a **ring** scotoma. A **positive** scotoma is one which brings itself to the notice of the patient, a **negative** scotoma is one of which he is unconscious. A positive paracentral or peripheral scotoma is most likely to be caused by an opacity in one of the refractive media, for the simple absence of vision in an island of the retina which does not include the fovea is not apt to be noticed subjectively, unless it is quite large, although there may be a certain amount of consciousness of its presence when the retinal pigment and the choroid are affected, but a central scotoma is sure to attract attention through the impairment of the central vision. Negative paracentral and peripheral scotomata usually can be referred to lesions in the retina or choroid, though occasionally they are met with in disease of the central nervous system.

The Blind Spot

A round, or elliptical, absolute, negative scotoma is present in the field of every eye, between twelve and eighteen degrees to the outer side of and about three degrees below the point of fixation, corresponding to the place of entrance of the optic nerve. This is known as Mariotte's blind spot. Its presence proves that the fibers of the optic nerve cannot be stimulated by light until after they have entered the retina. We find it enlarged, and perhaps misshapen, when medullated nerve fibers, conus, or posterior staphyloma is present, as well as in choked disk, in well marked neuroretinitis, and in glaucoma, but the principal diagnostic value of an enlargement of the blind spot is found in the cases in which it is symptomatic of a purulent inflammation of the posterior accessory sinuses.

Central Scotoma

When an object is seen better if it is a little to one side of the point of fixation the patient has a central scotoma. This may be relative, perhaps for red and green only, or it may be absolute, and it may be present in one eye or in both. If the patient states that objects appear to be distorted, or to be smaller than they really are, we shall probably find a central retinitis, a central choroiditis, or possibly a hemorrhage in the fovea centralis. A large, absolute central scotoma in one eye may be caused by a hole in the macula. Most cases of central scotoma are caused by disease of the papillomacular bundle of fibers of the optic nerve, and then usually, though not invariably, they are bilateral. As a general rule we may say that a central scotoma of one eye alone is due to a local lesion at the fovea, while one that is present in both eyes is indicative of disease of the papillomacular bundle, but this is a rule to which there are quite a number of exceptions. An opacity in any of the refractive media that happens to lie in the visual axis of the eye produces a central scotoma, and this may be present in either one or both of the eyes.

Ring Scotoma

We do not know much about ring scotomata, and the use to which they can be put in making a diagnosis, further than that most cases occur in connection with pigmentary degeneration of the retina, that

they are met with sometimes in choroiditis due to hereditary syphilis, as well as in diseases of the posterior ciliary vessels, and that they have been found in cases of diabetes, and perhaps in other diseases.

Scintillating Scotoma

When a patient describes the appearance of a positive central or paracentral scotoma in each eye, in the form of either a dark spot or a bright light, which enlarges until it has covered symmetrical portions of the two fields, while flickering bright points, or zigzag lines flash about in these areas, and these scotomata last from fifteen to thirty minutes before they recede, we say he has a scintillating scotoma. As a rule, these attacks are accompanied by vertigo, and are followed by pain in one side of the head, nausea, and perhaps vomiting, the usual symptoms of migraine, a disease to which this is so closely related that sometimes is is called **ophthalmic migraine.** Another name by which it is known is **partial fugacious amaurosis.** Sometimes the scotoma is not absolute, but forms a scintillating cloud before the eyes. It is said to have occurred in one eye alone, but such a case is uncommon.

The scotoma usually takes the form of a homonymous hemianopsia, but *Swanzy* states that "this defect may exist as symmetrical scotomata, complete or partial homonymous hemianopsia, or even horizontal hemianopsia. In some cases the scintillations may be absent, while in others the attack of migraine does not follow." He also says that it occurs most frequently in persons who are intellectually active, and that attacks have been known to be brought on by long continued reading, fatigue, and hunger.

The ætiology of this trouble is obscure. It is possible that the attacks are caused by an anæmia of the occipital lobes which is brought about by some circulatory disturbance, the nature of which is unknown. As a rule it does not indicate the presence of any serious cerebral lesion, though possibly it may in cases in which it is of frequent occurrence, and is associated with other symptoms.

HEMIANOPSIA

Occasionally a patient tells us that when he is looking straight forward the right or the left side of the field is dark, or that everything above or below the horizontal plane is obscured, but more commonly we discover an obscuration of a half field during the examination

of a patient who has symptoms of intracranial trouble. Such a phenomenon in only one eye suggests the presence of an intraocular lesion, such as a detachment of the retina, or a large hemorrhage, which interferes with the vision of one part of the retina, and we can imagine that such a lesion might occur symmetrically in both eyes so as to simulate bilateral hemianopsia rather closely. Glaucoma sometimes contracts the nasal part of the field quite decidedly, and when this disease is far advanced the rare binasal hemianopsia may be simulated to a certain degree, the more so as we sometimes find more or less of a concentric contraction of the preserved part of the field in hemianopsia, but the differentiation is easy because all lesions and diseases of the eye itself that can produce such a condition are gross and detected readily.

The **distinctive features** of hemianopsia are a more or less complete blindness of approximately one half of the visual field, delimited by a fairly straight line, with no ocular lesion or disease that can account for it. This condition almost invariably is present in both eyes, very rarely occurs in one alone, and it indicates that the corresponding parts of the retinæ have lost their function. This loss on the part of the retinæ is called **hemiopia.** The defect is said to be complete when it involves the entire half of the field, to be incomplete when a portion of the affected half is not involved. It is absolute when there is no perception of light in the area, is relative when light can be perceived but form and color cannot. The defect may be for color alone, when we call it **hemiachromatopsia.**

Hemianopsia is divided into **vertical** and **horizontal,** in accordance with the direction of the line which separates the darkened from the preserved halves of the field. Vertical hemianopsia is subdivided into ***bitemporal,*** in which both temporal halves of the fields are wanting; ***binasal,*** when both nasal halves are gone; and ***right*** or ***left homonymous,*** in which both right, or both left halves of the two fields are darkened. The homonymous is by far the most common form. The dividing line may be perfectly straight and pass through the point of fixation, but in most cases it makes a bend, or an angle, so as to pass around this point and leave it in the preserved portion of the field. Several cases of double homonymous hemianopsia have been reported in which a small central field of vision was preserved in this way. The reason why this central point is spared in some cases and not in others is not yet known with certainty, though theories have been advanced in explanation; it is

probable that there are anastomosing nerve fibers about the macula, and possibly there is a separate center in the brain for this part of the retina.

Hemianopsia always indicates a **lesion in the brain** that is so situated as to compromise the nuclei or fibers of the optic nerve, so when it is taken in connection with other symptoms it is of great value in localization. Bitemporal and binasal hemianopsia locate the lesion at the chiasm, as this is the only place where the fibers that pass to the temporal or nasal halves of the retinæ can be affected simultaneously. Horizontal hemianopsia, when present in both eyes, must be referred to an involvement of the upper or the lower fibers at the chiasm, to a lesion that affects both nerves simultaneously between the chiasm and the optic foramina, or to one that affects the upper or lower parts of both optic tracts in the same way.

A lesion that presses from below or above on the optic nerve of one eye may cause a horizontal hemianopsia of that eye alone, and a double lesion in front of the chiasm may cause a loss of the upper half field of one eye, and one of the lower half field of the other. It would seem as though a similar encroachment upon an optic tract from below or above, back of the chiasm, would produce a homonymous quadrant defect of the fields, which would probably be accompanied by other symptoms indicative of disease at the base of the brain. A similar quadrant hemianopsia, that is not attended by motor or sensory symptoms, can reasonably be referred to a lesion confined to one part of the cuneus, while if it is attended by hemiplegia and hemianæsthesia the lesion is more likely to be beneath the cortex of the occipital lobe. Homonymous hemianopsia indicates that the conductivity of the nerve fibers which belong to one optic tract has been interrupted somewhere back of the chiasm, to and including the cuneus. Lesions along this lengthy stretch are likely to induce other symptoms, which vary in individual cases, and pertain not only to the eyes, but also to the mind, and to the sensory and motor functions of various parts of the body. By means of these symptoms we are able to locate the trouble first within a rather large area, and then by the relative predominance of certain ones we can sometimes determine its situation with a fair approach to accuracy. The majority of these lesions are tumors, which are apt to be attended by choked disk, but this symptom alone does not help us much.

A right homonymous hemianopsia shows the lesion to be on the

left side of the brain, and, conversely, the trouble is on the right side when the left half fields of vision are obscured. If the hemianopsia is attended by no motor, sensory, or mental symptoms we place the lesion in or about the cuneus. If the patient has no motor or sensory symptoms, but is unable to recognize, or to name correctly, objects when he sees them, although he is able to do both by means of his other senses, or if he has such psychic troubles as alexia, dyslexia, or color amnesia, we incline to locate the lesion outside of the cuneus in the optic radiations. As sensory disturbances appear we locate it farther and farther forward, until, when there is hemianæsthesia of one side of the body, perhaps with ataxic movements, but with no distinct paralysis, we place it in the optic thalamus. The appearance of motor symptoms guides us still farther forward until, when the patient has both hemianæsthesia and hemiplegia, we look for it in the posterior part of the internal capsule. From this point forward the anæsthesia diminishes, and hemiplegia with little or no diminution of the sensitiveness of the skin suggests the region of the middle and anterior lobes of the brain. An absolute hemianopsia aids little of itself in the localization of the lesion, as it may be produced by one located in the cortex or elsewhere in the brain, but a relative hemianopsia we expect to find to be produced by a lesion in the cortex. Sometimes we can determine whether the trouble is in front of or behind the geniculate bodies by means of **Wernicke's test of the pupillary reaction.** This test must be made in a dark room with only just enough illumination to enable us to perceive whether any movement takes place in the pupil or not. A small beam of light is cast into the eye in such a way that it will fall upon the hemiopic part of the retina, and then turned so as to fall on the functionating part as nearly as possible at the same distance from the fovea. If the pupil does not contract when the light falls on the hemiopic portion, but does when it falls elsewhere on the retina, we know that the lesion is situated in front of the geniculate body; while if the reaction of the pupil is the same whenever the beam of light falls upon any part of the retina that is at a certain distance from the fovea, the lesion is behind the geniculate body. This test is one which is difficult to make, and does not give us as much certainty as could be desired, because light has a strong tendency to become diffused after it has entered the eye, and so to affect all parts of the retina equally. We have to combat this tendency by using as small a beam of light as will excite a reaction when cast upon the healthy

retina, and then be careful to throw it in exactly the right direction. Some cases have been met with in which the light when cast on the hemiopic side induced a slighter reaction of the pupil than when it fell on the other side, but the effect of diffusion would seem to be hard to exclude in such a case. The test can be used only when the hemianopsia is absolute. A concentric contraction of the preserved portion of the field does not necessarily indicate that the lesion is situated in the cortex, but it does so rather commonly.

CHAPTER XIX

Headache, Neuralgia, and Eyestrain

The subjective symptom which, more than any other, leads a patient to seek the assistance of a physician is **pain.** In nearly every case this is the one that predominates in his mind and most urgently demands relief, while sometimes it is so intense as to distract his attention from any other symptom with which it may be associated, even when under other circumstances they would be sources of great anxiety. The pain may be so great in a case of acute glaucoma as to cause him to fail to notice that he can no longer see with the eye until the fact is revealed in our examination. Yet there is no more unreliable guide to a diagnosis than pain, especially when it affects the head. When confined to a single organ it is called toothache, earache, eyeache, etc., and, as a rule, it indicates local trouble, but occasionally it is reflex and is induced by trouble somewhere else. We meet with cases of earache that are caused by carious teeth, with cases of eyeache that are due to indigestion, and toothache can likewise depend on a lesion in the eye. This fact needs to be borne in mind, for we should search out the source of a reflex pain in an organ which appears to be healthy. All reflex pain does not come from the eye, but when an aching tooth is sound, and a roentgenogram reveals no collection of pus at its root, it is well to test the tension of the eye on the same side, for there have been cases in which a perfectly normal tooth has been pulled in a vain endeavor to do away with a pain caused by glaucoma.

All other pains in and about the head commonly are grouped together under the term **headache.** Such a pain may be slight or severe, acute or chronic, superficial or deep seated, transitory or constant. Sometimes it is located with sufficient definiteness in a certain part so that we can speak of it as frontal, occipital, of the vertex, or through the temples, at other times it is generalized. We may try to describe its character by such terms as throbbing, stabbing, sharp, or dull, but none of these distinctions are of great use to us in diagnosis, in the majority of cases. A headache may depend

on functional or organic disorders of almost every organ in the body, on abnormal conditions of the blood, or of the blood supply to the brain, and on toxic conditions. This brief review suffices to show that although headache is a frequent accompaniment of a great many diseases of the eye, and forms one of the most conspicuous symptoms of eyestrain, we cannot expect to find its cause in the eye in more than a rather small proportion of cases.

Certain forms of headache are known by distinctive names. One that is confined to one side of the head is called **hemicrania.** A paroxysmal hemicrania that, as a rule, is ushered in by an aura and terminates with nausea, is known as **migraine, megrim,** or **sick headache.** This is rather apt to be associated with such ocular symptoms as pareses of the extrinsic muscles, or an affection of the optic nerve, and then we regard it as symptomatic of some functional or organic trouble in the brain. We seldom if ever discover the cause of a migraine in any disease of the eye, and though it is possible for it to be a symptom of eyestrain, we do not commonly find it to be such.

Facial Neuralgia

Although it may be claimed that all pain is neuralgic, as in every case a sensory nerve, or its filaments, is irritated in some manner, it is convenient to designate as neuralgia any pain that occurs in, and is confined to the area of distribution of, a sensory nerve. A pain, which usually is paroxysmal, that is confined to the area of distribution of the trigeminus, or of one or more of its branches, is known as **facial** or **trigeminal neuralgia.** When it affects only the region supplied by the ophthalmic branch we speak of it sometimes as **supraorbital neuralgia.** The condition is one which is easy to recognize when a patient complains of paroxysms of acute pain over certain regions of the face and scalp, and we find the skin over these regions to be hypersensitive, but this does not constitute a diagnosis; we have yet to learn its cause, for in nearly if not quite every case neuralgia is a symptom, and not a disease. The pain may be mild, moderately, or extremely severe, and in the last case it may radiate over the occiput down to the nape of the neck, or even to the shoulders. The paroxysms may be of very short duration, or may last for hours, and the intervals between them vary a great deal in length, but during these intervals we are apt to find the skin more or less hyperæsthetic.

Facial neuralgia, involving any or all of the branches of the trigeminus, may be caused by catching cold, by an injury in which the nerve is implicated, by general diseases, and by toxic conditions, as well as by affections of the eye, and of the orbit. We meet with it in the secondary stage of syphilis, in malaria, in chronic alcoholism, in gout, in diabetes, in many lesions of the brain, in affections of the nose, of the frontal sinus, and of the middle ear, and it may be the first symptom presented by some intracerebral disease, or by an attack of herpes zoster, so, in a very large proportion of the cases, we cannot expect to find the cause in the eye. Yet in quite a number of ocular diseases it forms a prominent symptom, and a **very severe attack,** particularly of supraorbital neuralgia, may lead both the patient and the physician to overlook symptoms which would be noticed immediately if it were milder. In such a case it takes but a moment to glance over the tissues of the anterior segment of the eye, and to test the tension with the fingers, but this will suffice to tell us whether any such inflammation is present as a keratitis, an iritis, or an acute glaucoma, while at the same time our attention will be drawn to any signs of trouble that may exist in the orbit, like a periostitis. Neuralgia is excited by the eye only when some of the terminal filaments of the trigeminus are affected, and therefore is not likely to be a manifestation of deeply seated lesions, except when these have induced a secondary glaucoma, so this rather cursory examination of the eye is sufficient when the patient is suffering from a very severe attack of facial neuralgia.

A different condition confronts us when the patient gives a history of repeated **mild** or **moderate attacks** of facial neuralgia, especially when the intervals between them have been constantly growing shorter, as it is suggestive of a possible chronic inflammatory glaucoma. Again in these cases the neuralgia is usually, but not necessarily, supraorbital. We should test the vision and examine the papilla in search of a glaucomatous excavation. If the tension does not seem to be above the normal limit we should not be content with the test with our fingers, but measure it with the tonometer, repeating the examination several times if necessary. Above all we should seek to examine the eyes for slight symptoms during an attack of the neuralgia. When no pathological lesions can be detected in the eye we should investigate its refractive and muscular conditions, though it can scarcely be said to be likely for a neuralgia to be one of the symptoms of eyestrain.

EYESTRAIN

The great majority of our patients present no serious inflammation or disease of their eyes, but give us histories of certain rather indefinite, though annoying symptoms, such as headache, dizziness, sickness at the stomach, which may or may not be associated with a pain, or a tired, gritty, itchy, burning, or sleepy feeling in the eyes, or with either a constant or a transient blurring of the vision. Some of them are anæmic, others have a hyperæmia of the conjunctiva, perhaps associated with a redness of the margins of the lids, and still others have no such symptoms. We class all of these patients together as victims of eyestrain, but this term in no way implies a diagnosis. In so far as it seems to do so it is a misnomer, for while in many cases we can trace the origin of the symptoms to a fault in the refractive or muscular condition of the eyes, in others the cause finally is revealed somewhere else in the body. There is nothing about the symptoms themselves to guide us with any certainty, for even when they are confined to the eye alone, the cause may be situated in some other organ. There is no department of ophthalmic work in which the eye surgeon needs to be more deeply versed in general medical knowledge than in this, the search for the cause of asthenopia, or of eyestrain, and yet this is contrary to current opinion. The fitting of glasses, which in most cases should be held to be synonymous with this search, is entrusted, not only by the laity, but by physicians as well, to persons who admittedly have no medical knowledge, and it may be that we ourselves are responsible to a certain extent for the current opinion. The very fact that these patients form the bulk of our practice acts as an incentive to yield to the temptation to hurry through with them, and to ascribe their troubles to such faults as we may happen to find, or in which we chance to be particularly interested.

Donders described as **asthenopia** a condition in which the patients complain of more or less inability to use their eyes continuously for near work without the occurrence of a feeling of fatigue, tension, or strain in, about, or especially above the eyes, which may be painful, burn, sting, or simply feel uncomfortable, associated at times with blepharospasm, lacrimation, and photophobia, and often with more or less blurring of the sight, although the eyes themselves seem to be perfectly normal, their vision to be good, and their motility to present no obvious defect.

We use the term **eyestrain** in a broader sense, so as to include not only the symptoms of asthenopia, which pertain particularly to the eye, but also those of reflex irritation of the nervous system. Of the latter headache is the most common, while dizziness, loss of appetite, indigestion, nausea, vomiting, and signs of nervous irritability that range all the way from peevishness down to insomnia, mental depression, and nervous breakdown, and shown sometimes by muscular twitchings, are very frequent. According to some writers chorea and epilepsy should be added, but it is only in rare cases that these diseases are to be ascribed correctly to eyestrain. These symptoms do not form a syndrome. The presence of any one, or of any group of them indicates that the nervous system is irritated by trouble somewhere in the body. They are well known to practitioners in other departments of medicine, by whom they are spoken of usually as associated nervous symptoms, and we speak of them as signs of eyestrain simply because in many cases they are dependent on ocular faults.

It is natural and proper that the organ which presents the most striking symptoms should receive attention first. Probably it is true that the trouble is to be found most frequently in the stomach when the gastric symptoms are most marked, in the central nervous system not only in such diseases as chorea and epilepsy, but also in cases of nervous exhaustion, and in the eye when the asthenopic symptoms predominate. Although this rule is by no means a positive guide, it is of much help to us indirectly, as in many cases it has led to the exclusion of faults in other organs by other physicians before the patients come under our observation. The important point we must remember is that the site of the principal symptoms is not necessarily the site of the trouble. ***Exactly the same group of symptoms*** may be caused by over work or over worry in one case, by gastric disturbance in a second, by a refractive or muscular fault of the eyes in a third, and by a lesion in the nose or some other organ in a fourth. Furthermore, these symptoms of reflex irritation of the nervous system may depend on the combined effects of causes situated in two or more organs, so that partial, but not complete relief may be given when the fault in one is rectified, and the real cause may be located in an organ that exhibits no signs of trouble whatever. We know that the eyes are at fault in many cases in which they look and feel perfectly well, we know that the symptoms can be excited by pressure in the nose when the patient

has never been made aware of any trouble there, and we are not justified in the assumption that among all of the organs in the human body the eyes and the nose are unique in this respect. In view of these facts we should hesitate to speak dogmatically, we should admit rather that in most cases we have to be content with a tentative diagnosis, at least until after we have had a chance to observe the effects of treatment directed to the abnormal condition we have found. When the effect of such treatment is to produce a complete and permanent relief to the symptoms it is reasonable to suppose that the diagnosis was correct. We may say positively that it was correct when the treatment was a logical one which has secured success in the hands of many different surgeons. But when we consider the powerful effects of suggestion exerted by a strong personality, even when the physician does not exert it consciously, in connection with the facts that these symptoms are excited in many cases by functional derangements rather than by organic changes, and that such cases are peculiarly susceptible to suggestion, the results obtained by unusual, or not perfectly logical treatment in the hands of a few surgeons of unquestioned probity but very strong personality, are apt to leave doubts in our minds as to the accuracy of the diagnoses. These doubts are accentuated when other trained surgeons fail to do as well.

Each patient should be studied individually. We should take into account his age, his history, his occupation, his physical and nervous condition, and often consider the relations that the symptoms manifest toward one another. We should not be content with our refractive and muscular findings, which must be determined slowly if we would be accurate, but we should start with the understanding that the symptoms of which the patient complains may be caused by an error of refraction, by an imbalance of the ocular muscles, or by trouble elsewhere in the body; and that there is no refractive error, no heterophoria, and no outside condition that necessarily is the cause in any particular case. The difficulties of diagnosis in this class of cases are not due to the fact that it is hard to measure with accuracy refractive errors, or any of the various forms of heterophoria, as much as they are to the multiplicity of possible causes.

Eyestrain Due to Refractive Errors

The most prolific source of eyestrain that comes under the observation of the ophthalmologist is a fault in the refraction, so the first thing for us to do is to ascertain whether the eyes are optically perfect in construction or not. We place the patient in front of a well lighted chart of testtypes of various sizes so graded that each should be distinctly visible at a certain known number of feet or meters, cover one eye and have him read the smallest characters that he can. To transform this subjective symptom into an objective sign, which we can appreciate and utilize, we indicate it by a fraction in which the number of the feet or meters from the patient to the chart is used as the numerator, and the number of feet or meters at which it has been calculated that the smallest characters he reads should have been recognized is taken as the denominator. If he can read no smaller type than that which should be read at thirty feet, or nine meters, when he is twenty feet, or six meters, away from them, we record his vision as 20/30, or 6/9. If he reads those calculated to be read at ten feet, or three meters, we write 20/10, or 6/3. Then we test the other eye in the same way. This gives us the uncorrected vision of each eye, but not the actual visual acuteness, which may perhaps be improved by the correction of a refractive error. When this has been done for each eye separately we have learned the real vision of each. The standard for visual acuteness that has been adopted is far from satisfactory, and many efforts have been made to secure a better one, but it seems rather doubtful if a uniform standard can be devised, because the acuteness of vision varies physiologically in different persons, and appears to be modified by the demands made upon it in daily life. As a rule we may accept the standard of 20/20 as the minimum of perfect vision, ascertain the best possible in each individual case, and if it cannot be brought up to this minimum it is incumbent upon us to learn the reason why. We call to our aid the ophthalmometer to determine whether the curvature of the cornea is normal, the ophthalmoscope and the retinoscope to measure the refraction objectively in the different meridians of the eye, yet the case of test lenses must remain the court of last resort until some method is devised by which we can compute objectively the normal activity of the ciliary muscle in the individual. Ophthalmic literature is full of instructions how to use these instruments for the measurement of refraction, each writer

advocating a method that seems to him to give better results than any other. Some employ cycloplegics as a routine measure, others use them only in selected cases, and it is probable that each method is the one best suited to the person who advocates it; that is, by its means he is enabled to make fewer mistakes than he would if he used any other. Yet none of these methods are perfect. Every eye surgeon knows that he has been successful in giving comfort to a large majority of his patients, and that he has failed to do so in a minority, but he has no data from which he is able to form even a reasonable conjecture how many of his failures were due to a wrong measurement of the refractive error, and how many to a wrong diagnosis in ascribing the symptoms to the refractive error, because most of those patients went elsewhere for relief. He only knows from his successes where others have failed that no one always measures refractive errors rightly. Without entering into the controversy as to the merits and demerits of the various ways of determining the refraction of the eye, it will suffice to say that the fundamental principles are to be obtained from any one of a great number of text-books, some devoted wholly to this subject, and that it is best to adopt some one method with a view to becoming as nearly perfect as possible in its application, for in that way we shall make the fewest mistakes.

We shall examine very few eyes in which we are not able to find an error of refraction, but the ***positive demonstration of such an error is not sufficient.*** One of the most difficult things for us to appreciate is the widely varying effects produced by the same error of refraction upon different individuals. In the great majority it produces no effect of which the person is conscious, unless it is so great as to impair the vision. Even when it does cause trouble the eyes are so very tolerant that they are made perfectly comfortable by an approximate correction, and remain so when the mountings are bent, when a spherical lens is tilted so as to produce an astigmatic effect, or when the axis of a cylindrical is altered five, ten, or even fifteen degrees. Persons with such tolerant eyes are easily satisfied, as might naturally be expected. A comparatively small number of eyes are intolerant to even a very slight error of refraction, and to a slight variation of the correcting lens from its proper position. These demand accuracy not only in the measurement of the refractive error, but also in the adjustment of the correcting lenses, which should be so placed in a plane perpendicular

to the line of vision that each eye looks through the optical center of its own lens, while, if the correction is cylindrical, the axis is exactly right. Faults in which the optical center does not coincide with the midpoint, in which the visual lines pass to one side of, above, or below the optical center, and in which the axes vary some degrees from their correct position, are very common in glasses as they come from the opticians, who have learned by experience that slight faults of this nature are not noticed by the great majority of people, and see no profit to themselves from the expenditure of the time and money necessary to secure accuracy. A certain proportion of our failures to give relief is to be ascribed to such faults as these, so we need to investigate each pair of glasses furnished as an essential for success not only in treatment, but also in diagnosis.

After we have measured the refractive error we can obtain positive evidence that we have discovered the source of trouble in many patients, though not in all, by *having them wear the properly adjusted correction for a short time.* If the symptoms of eyestrain are completely, or even partially relieved, we have reason to feel confident that the corrected error is at least one of the sources of the trouble, while if no relief is felt we are guided to search elsewhere. When the patient is made comfortable by the test lenses, but the same comfort is not secured by the glasses provided by the optician, the probability is that the latter are at fault either in the situations of the optical centers, in the positions of the axes, or in the adjustment of the lenses before the eyes. When no relief is given by the test lenses the probability is that the source of trouble is elsewhere, but sometimes we eventually find the refractive error to be if not the primary, at least a contributory cause.

At this point mention may be made of two widely prevalent erroneous ideas that we should do our best to controvert. We meet with many people who delay and avoid the wearing of glasses at the expense of much trouble, annoyance, and sometimes injury to the eyes, because they have the idea that by so doing they will keep these organs in a better condition than they would be in if glasses were worn continually. The bugbear of becoming so habituated to their glasses that they cannot do without them is a constant source of worry. These people need to be assured that the constant strain upon their eyes when they refuse to wear a needed correction tends to weaken rather than to strengthen these organs; that no one can become habituated to glasses that they do not need so as to make

their aid a necessity, and that anyone can at any time cease to wear his needed glasses if he will do what they are doing—accept the discomfort and trouble from which they are freed by such aid. Another class, composed mainly of young ladies, decline to wear glasses because they think the latter do not accord with their own particular styles of beauty. They accept present discomfort and possible danger in the future with a cheerfulness we envy, and welcome the suggestions of the former class that they are conserving their eyes by not following directions. We can do little for these beyond presenting the facts as plainly as possible. The other erroneous idea is that the wearing of glasses when they are not needed tends to conserve the eyes. Parents sometimes bring children who are in perfect health and present no symptoms whatever, to have their eyes examined, in order to forestall future trouble. Almost invariably it is possible to prescribe glasses that can be worn, but the wearing of them is useless when they neither alleviate symptoms, nor improve vision; they neither conserve the vision, nor protect the eyes from ill, except that in some cases they may help to guard against the development of school myopia.

Presbyopia

When a patient 45 or more years old tells us of an inability to use his eyes for reading, sewing, or other fine close work, we make a positive diagnosis of presbyopia, because we know that the near point recedes with the advance of age, and that usually in the fifth decade of life it gets so far away as to render near work impracticable. This is a purely physiological condition, but the claim of some writers that it never gives rise to symptoms of eyestrain is much too broad. It is true that in most cases of uncomplicated presbyopia an approximate correction is satisfactory to the person, who is quite as well pleased with the results obtained by picking out glasses for himself, or by having them chosen by an optician, as by having them fitted accurately through a scientific examination, but occasionally the eyes rebel. An example of this nature was furnished by a gentleman 46 years old, who complained that the + 1 D lenses which he wore for reading strained his eyes unbearably, and was given perfect comfort by a pair of + 1.25. Presbyopes who complain of eyestrain are quite apt to have other refractive errors, or some form of muscular imbalance, so the examination of the eyes should

always be thorough, but we must beware of certainty in ascribing the symptoms to these faults. If the correction of the presbyopia gives complete relief to the symptoms the diagnosis of eyestrain from presbyopia is positive, no matter what other abnormalities may be present, and it is worth noting in this connection that the simultaneous correction of an astigmatism, which has never given rise to symptoms, may plunge an elderly patient into trouble that can be allayed only by the removal of the cylindrical correction.

The rule is that presbyopia becomes manifest about the age of forty-five, and that its presence then can be demonstrated after the correction of a hypermetropia or a myopia, just the same as in emmetropia, although it often seems to appear earlier in hypermetropia, and sometimes not at all in myopia, when these conditions are uncorrected. The reason of the last is that the far point of the eye is at about the reading distance. The cause is supposed to be a sclerosis of the lens, which begins in early life and about this age reaches a point at which it has deprived the lens of so much of its ability to change its shape readily with the contraction of the ciliary muscle as to reduce the range of accommodation enough to make near work difficult. The possible exceptions to be met with are too few and too uncertain to invalidate this rule in the least. Cases occur in which the accommodation is sluggish in its action, so that the symptoms appear sooner than we expect them, and I have seen two cases in which only a single diopter of correction was needed at sixty-seven and sixty-eight, although both patients assured me that they had always enjoyed sharp vision for distance, had never needed glasses before, and the refraction in both was slightly hypermetropic. This history and the low degree of presbyopia together seemed to indicate a late onset of the latter, though the possibility of a former slight myopia could not be excluded objectively. It is by no means unusual for the presbyopia to be unequal in the two eyes, but whether this inequality is to be referred to differences in the lenses or in the ciliary muscles is unknown.

Hypermetropia

When a person has equally as good vision with a convex glass as without it, and when his vision is improved by such a glass, he has hypermetropia. If he suffers from eyestrain this may or may not be the cause, and our problem then is not simply to measure the de-

gree of the hypermetropia, but **to ascertain whether it is associated with an *abnormal* strain** upon the accommodation that is responsible for the suffering.

We must not assume that hypermetropia is of itself abnormal, even though we commonly speak of it as an error of refraction, nor that the tonic contraction of the ciliary muscle which accompanies it is physiologically wrong. We are obliged to concede both to be physiological rather than pathological when we take into account the development of children's eyes, and the fact that the great majority of the people who have never had any reason to think that there was anything the matter with their eyes have a certain degree of hypermetropia. With few if any exceptions babies are born with eyes that are too short for convenient use in the seeing of either near or distant objects; these eyes lengthen, rather rapidly at first, then more and more slowly as the resisting power of the sclera increases, until the process comes to a standstill. If this process of lengthening ceases when the eye is just long enough to allow parallel rays of light that fall upon the cornea to come to a focal point on the fovea without any assistance on the part of the ciliary muscle, the condition is one of emmetropia, which is commonly accepted as the normal, or at least as the ideal refraction of the eye. Perfect emmetropia is very far from common. If the eye has become lengthened beyond this point, so that parallel rays cross before they reach the retina, a condition of axial myopia has been produced, over which the ciliary muscle has no control whatever. If the cessation of the process occurred before the eye had attained its ideal length, the parallel rays of light that enter can be focussed on the fovea only through the active intervention of the ciliary muscle, if at all, because they tend to meet behind the retina, and the condition is that of hypermetropia. The presence of good vision in a hypermetropic eye when it is looking at a distant object proves that the ciliary muscle is in a state of activity which we call its **tone,** or tonic contraction. This tone differs in no way from the normal tension to be observed in all healthy tissues, is called by the same name, and should not be looked upon as abnormal. Although theoretically we should find the best vision in eyes that are emmetropic, my observation has been that the greatest acuteness of vision is to be met with in eyes that are slightly hypermetropic with a normal tone of the ciliary muscle, and the best explanation of this that has occurred to me is that possibly the constant call to action on the part of the latter

enables it to make a finer adjustment of the accommodation than is made when its relaxation is complete at times. It does not seem right to speak of eyes as faulty that have perfect vision, give their owners no trouble, and are in a condition which is known to be physiologically normal in all eyes during early childhood and remains in most of them throughout life. At the same time the condition is one that gives rise to eyestrain occasionally through an abnormal variation of the activity of the ciliary muscle from its normal tone.

We divide hypermetropia into the **manifest,** or the portion represented by the strongest convex lens through which the eye retains perfectly distinct distant vision while the accommodation is intact, and the **latent,** which is hidden by the contraction of the ciliary muscle and is revealed when this muscle is paralyzed, either by disease, traumatism, or drugs. The manifest and the latent together constitute the **total** hypermetropia. The ratio of manifest to latent hypermetropia varies not only with the amount of the total, but also with the age of the patient, and with the strength or weakness of the general muscular tone throughout the body, so it cannot be stated in any definite figures which are applicable to individuals. It is generally true in a person who enjoys ordinarily good health and strength and has a moderate degree of hypermetropia, that the latter is almost wholly latent in early childhood, that the manifest steadily increases at the expense of the latent until the two are about equal at the age of twenty-five, and that the latent has nearly disappeared at forty. About the last age it happens very often that a patient complains of difficulty in reading, writing, or other near work, sometimes that his distant vision also has become more or less indistinct, but of no other trouble, and then we almost invariably find that these symptoms are relieved perfectly by glasses through which his distant vision is perfect. The symptoms are exactly the same as those of presbyopia, but they are caused wholly by the hypermetropia. When the degree of hypermetropia is so great that the ciliary muscle is unequal to the task of focussing on the fovea the rays of light which enter the eye, the vision is subnormal, but can be brought up to or above the accepted standard by means of convex lenses. Such a degree of hypermetropia seldom induces symptoms of eyestrain, it simply renders distant vision indistinct, perhaps because the ciliary muscle refuses to attempt to maintain habitually a sufficient degree of contraction.

As long as the **normal tone** of the ciliary muscle is sufficient to

overcome easily the degree of hypermetropia present the person is absolutely unaware of any effort, and is inclined to boast of his excellent eyes, but when the tone is modified so that the contraction of the muscle is less or greater than normal, symptoms of eyestrain are apt to appear. Possibly these symptoms are likely to be produced when the normal tone of the ciliary muscle is habitually taxed to its utmost, but we are not sure of this. As age advances it becomes more and more difficult for this muscle to overcome the hypermetropia, and when finally the vision has become subnormal we know that there was a time when perfect distant vision was maintained only by the full force of the normal tone, yet many patients who have difficulty in seeing objects distinctly either in the distance or near at hand, tell us of the fine sight they formerly possessed and of its loss, but make no mention of any symptoms that we can refer to eyestrain which appeared at, or a little before the time when their vision began to fail, while other patients in the same condition do tell us of a past possible eyestrain at about that time, when we can find no evidence of muscular weakness, or of spasm. Such a history is inconclusive because the symptoms might have been caused by some trouble elsewhere, which was in no way connected with the eyes.

We know that a ciliary muscle, which has proved itself fully competent to correct the hypermetropia during good health, is apt to become unable to do so when all of the muscles of the body have been weakened, and we often see symptoms of eyestrain appear when the eyes are taxed during convalescence from sickness, to pass away as the muscular system regains its normal tone. When we measure carefully the manifest hypermetropia of a young adult who is suffering from eyestrain by slowly increasing the strength of the convex glasses by which it is corrected, it frequently happens that we find the manifest to be nearly or quite equal to the total as determined under atropine, instead of maintaining about its normal ratio to the latent, and then almost invariably we find the tone of all of the muscles in the body to be below par. If the symptoms are relieved in such a case by convex lenses that correct the hypermetropia it seems to me that the diagnosis should be eyestrain due to **abnormal weakness of the accommodation** dependent on some cause outside of the eye which affects the musculature of the entire body, though ordinarily we speak of it as caused by the hypermetropia.

We meet with exactly the opposite condition in many cases in which the constant demand made upon the ciliary muscle has induced

a contraction that exceeds its normal tone. We call this **spasm of the accommodation** and find it to be a common cause of eyestrain. These cases form the great bulk of those in which the use of a cycloplegic is admittedly necessary. Sometimes we can secure a certain amount of relaxation by allowing the patients to look for a while through the convex lenses that correct the manifest and gradually increasing the strength of the glasses as the manifest increases, and, if we have perfect control over our own accommodation, we may be able to measure the total with the ophthalmoscope and so learn what proportion is latent. When we can do this we are apt to find that the latent seems to be greater than it should be in proportion to the manifest, when we take into account the age of the patient and the muscular tone of the body, or we may find an apparently myopic eye to be hypermetropic, but we have no reliable rule to guide us concerning the ratio of the manifest to the latent hypermetropia, and we are unable to be positive that we have unearthed all of the latent until we have used a cycloplegic. Less often we meet with cases in which the spasm of the accommodation is so strong and persistent that we can make out little if any variation from the manifest hypermetropia or myopia without a cycloplegic, and my experience is that the weaker cycloplegics are of little value in this condition. An example which is illustrative of quite a number is that of a young lady who had consulted several excellent ophthalmologists in search of relief from eyestrain, and had been adjudged by all of them to have not over a diopter of hypermetropia. The ophthalmoscope revealed no more, but atropine, which none of them had employed, disclosed four diopters in each eye, and subsequently I was told that the relief was complete. While the eye is under the influence of such a drug we have no means of determining the normal ratio of the manifest to the latent hypermetropia in that particular case, as an estimate made from the age and the general muscular tone is open to serious sources of error, so we have to wait until its effects have passed off before we can determine the true manifest. When the correction of this relieves the symptoms permanently we are certainly justified in saying that the eyestrain was due to the hypermetropia.

A complete relief to the symptoms while the eyes are under the influence of a cycloplegic leads us to feel pretty confident that we shall find the cause to be either hypermetropia or astigmatism, but it is not a sure sign. The eyestrain from muscular imbalance may be

noticeable only when the patient is engaged in near work, and the symptoms of nasal asthenopia are apt to be brought into prominence by the position of the head during near work, a position which is not likely to be taken while the eyes are incapacitated through paralysis of their ciliary muscles.

Symptoms of eyestrain due to this cause almost invariably require the full correction of the manifest hypermetropia for their relief, but occasionally we meet with a surprise. A gentleman who complained of eyestrain had been fitted elsewhere with glasses that seemed to be perfectly correct. He could read 20/15 through them with each eye, and no better vision could be obtained for distance, yet his symptoms were relieved completely by a reduction of half a diopter in the strength of each lens. A lady who had been wearing simple cylindricals without benefit to her eyestrain accepted for her best distant vision + 1 D spherical combined with + .50 cylindrical axis 90° over each eye, but secured relief only after the strength of the spherical correction had been reduced one half. Another curious case in this connection was that of a lady 46 years old who enjoyed perfect ease as long as she wore her correction for hypermetropia, except for the fact that she had to hold a book, or any near work, unpleasantly far from her eyes, but suffered from intense headache as the result of every attempt to correct her presbyopia.

Myopia

The great majority of cases of eyestrain met with in myopia will be found to be due to some other trouble, for it is doubtful if this refractive condition ever excites such symptoms, at least unless it is associated with astigmatism, and even then the symptoms are apt to be slight. Probably this is because no strong muscular effort is ever habitually required within the eye. When eyestrain is associated with an apparent myopia we should make sure that the latter has not been simulated by a powerful spasm of the accommodation, and the most certain way to do this is to instill a solution of atropine into the conjunctival sac. The spasm yields within a few days in the majority of cases, and then we almost always find the refraction to be hypermetropic. In exceptional cases the spasm is of some other origin, and I have grown to suspect this to be the case when it is unusually resistant to the effects of this drug. In one youth who apparently had about two diopters of myopia with some hyperexo-

phoria the ophthalmoscope revealed emmetropia, but the spasm did not yield until after the atropine had been used for some two months. In another youth who had apparently about the same degree of myopia with both the test lenses and the ophthalmoscope the severe symptoms of eyestrain led me to instill atropine for several weeks with no avail, and then to perform a partial tenotomy on his superior rectus in the hope of relieving the symptoms through a correction of from 2/3 to 3/4 of a degree of hyperphoria. To my surprise he read 20/20 with his naked eyes after the operation, and the refraction appeared to be emmetropic, although no change in the muscular condition could be detected two weeks later. In both of these cases the eyestrain seemed to depend on the spasm of the ciliary muscles in eyes that were at any rate not very hypermetropic. Whether it was caused in either or in both by the muscular imbalance is an open question.

We must not be misled by the fact that a spasm of the accommodation can simulate myopia in rather rare cases into thinking such a spasm to be present because we find the myopia to be slightly less when the eye is under the influence of a cycloplegic than when it is not. This is particularly true if the patient does not complain definitely of eyestrain. Such a variation is physiological, and is the measure of the tone of the ciliary muscle. Unless contraindicated by some other consideration the full amount of the myopia should be corrected, because the myopic eye tends to keep its health better when its ciliary muscle is called upon to perform its normal amount of work, and the patient then enjoys his best vision.

Astigmatism

Many cases of eyestrain are due to slight degrees of astigmatism, which may be either simple or compound, but almost invariably is hypermetropic. We obtain a fair estimate of the amount and the axis of the astigmatism from the curvature of the anterior surface of the cornea as shown by the ophthalmometer, and by means of retinoscopy when the eye is under the influence of a cycloplegic, but neither of these methods is infallible, both must be controlled by our findings with the test lenses and by the subjective sensations of the patient. Often when we place the lenses that correct the astigmatism before the eyes of the patient and have him take his attention away from the test card, we are gratified by the remark that the glasses

have relieved the headache completely, and then the diagnosis of eyestrain from astigmatism is made. Not so very infrequently we attain this result with cylindrical lenses that do not correspond exactly with our findings by any objective method, but are a little weaker or stronger, or have their axes a little different from what these findings would lead us to expect, and when we find this to be the case we know that we are dealing with an uncommonly intolerant eye, for most eyes accept an approximate correction readily, as has been mentioned already. After we have ascertained by our tests what we believe to be the correction of the refractive error we can allow the patient to **wear the correction** for a few minutes and note the effect produced on the symptoms. If the relief is marked, or if the patient is conscious of a steady, gradual improvement, we feel confident that we have made the diagnosis; if little or no relief is felt we modify the glasses slightly both in strength and position and give full opportunity for each change to produce relief if it can, but when none can be secured in this way it is well to investigate for other possible causes. In my experience, a correction of a refractive error which produces no effect on the symptoms of eyestrain at once is not very likely to succeed later, even though it may occasionally. Another fact worth bearing in mind is that sometimes when the patient has a low degree of compound hypermetropic astigmatism and is not relieved perfectly by the full correction, it may happen that a brilliant result can be obtained by eliminating the spherical portion of the correction, when we are justified in the conclusion that the eyestrain depends on the astigmatism alone.

EYESTRAIN DUE TO HETEROPHORIA

Very little has been written on the differentiation of heterophoria as the cause of eyestrain. Most writers seem to assume that the demonstration of a muscular imbalance is equivalent to the diagnosis, yet this can easily be proved not to be the case. We meet with all of the various forms of heterophoria in persons who show no signs of eyestrain, as well as in others in whom the symptoms can be traced to other sources, so that in any individual case the demonstration of an exophoria, an esophoria, a hyperphoria, or a cyclophoria, is that of a possible, but not necessarily of the actual cause, just the same as that of a refractive error, or of existing pressure within the nose. Heterophoria may be one of the symptoms induced by a mental or

physical factor that disorders the nervous equilibrium, and then is symptomatic of, rather than a cause of the trouble. Not infrequently it passes away, or at least fails to make its presence felt, when glasses that correct a refractive error are worn constantly. It sometimes appears in the course of diseases of the central nervous system, and I have seen several cases in which eyestrain with heterophoria seemed in the light of subsequent developments to have been symptomatic of derangements or diseases in the gastrointestinal tract, or in the genitourinary organs. This question of differentiation seems to me to deserve more attention than it has yet received, for there is no doubt that in an uncertain percentage of the cases heterophoria is the direct cause of eyestrain, and that some forms are more prone to excite these symptoms than are others. It is rather in the hope of stimulating investigation along this line, than of presenting anything final that the following points are propounded as of possible diagnostic value.

When the patient has found that he is able to relieve his symptoms of eyestrain and to continue his work in comfort by **closing one eye,** the source of the symptoms is located almost certainly in his eyes, and the chances are that it will be found in an imbalance of the muscles, though occasionally we will find it in a refractive error.

To be the cause of eyestrain a heterophoria must be an **anatomical entity and not an induced anomaly.** We can induce heterophoria artificially by exercises of the muscles with prisms, by wearing prismatic glasses, and by repeated tests of the muscles. About twenty years ago, while engaged in the study of the effects of prisms on my own eyes, I developed a hyperphoria that compelled me to wear a five degree prism base down over my right eye for a year and a half, which demonstrated how an artificial heterophoria could be produced. Another example was furnished by the patient alluded to who could disclose at will fifteen degrees of either exophoria or esophoria, but had orthophoria by the cover test. A heterophoria which has been induced in such a manner as this may appear to be real, so we should inquire into the possibility of an artificial production before we make our diagnosis. It does not seem to comport with common sense that a heterophoria which has been induced artificially should be considered the cause of antecedent symptoms.

The findings by all tests should show about the **same degree** of the **same variety** of heterophoria, and should they not agree the discrepancy should be explained before we assume that it is caused

by an anatomical fault. At the time I was obliged to wear a five degree prism base down, to relieve the annoyance produced by an artificial hyperphoria, my eyes were examined with the tropometer, which showed their rotations up and down to be perfect, while the rotation of one eye inward was imperfect; the perfect rotations up and down seemed to promise recovery from the hyperphoria, which took place later, and the imperfect rotation inward coincided with the findings by no other test.

The findings should **not fluctuate** greatly after short intervals of time. Marked fluctuations hardly seem to be consistent with a fixed error that is responsible for symptoms, and my attention has been attracted by such vacillations in several cases in which the heterophoria seemed to be symptomatic of an instability of the nervous system induced by over work, or by trouble in the abdominal organs. In quite a number it has seemed to be a question whether the symptoms of eyestrain with heterophoria did not form the first indication of kidney disease that was observed, while in others these symptoms developed after the presence of the disease had been recognized, but in all of them the fluctuations in the degree of the heterophoria were considerable, and to me at least were puzzling. This may be explained as simply a coincidence in the small number of cases that were observed, but when fluctuations are present it can do no harm to investigate the other organs of the body before we decide that the heterophoria is the cause of the eyestrain.

The prism convergence, the prism divergence, or the sursumvergence of the eyes **should show a fault** if the imbalance of the muscles is very marked. This is not necessarily so when the heterophoria is slight. We may consider these tests to be corroborative when the adduction is less than normal while the abduction is of full strength or excessive in a case of marked exophoria, or when the reverse is true in one of a high degree of esophoria. The sursumduction of the two eyes should be alike, and when a weakness of one corresponds to a hyperphoria it is confirmatory of the latter. When these tests show that the power of all of the muscles is subnormal it is well to consider whether we are not dealing with a general nervous or muscular weakness, which may be the origin of any anomaly of muscular balance present.

After we have gathered all of the information we can from the different tests already enumerated, including the one for cyclophoria, we have all of the data we can obtain from the muscles themselves,

and should exclude all of the other possible causes of eyestrain before we speak positively of the diagnosis. The final test in a doubtful case is the **result of treatment.** Permanent relief establishes the fact that the eyestrain was due to the heterophoria which has been corrected, provided we can exclude the effects of suggestion, but a temporary one does not. We may obtain a temporary, or a partial relief from the correction of a refractive error, or of an imbalance of the muscles, or of both, when the symptoms depend partially, but not wholly on these errors, and in such cases we should seek elsewhere for the cause, rather than try many times to secure an elusive balance of the muscles.

EYESTRAIN DUE TO CAUSES OUTSIDE OF THE EYE

The intimate relations that exist between the eye and all other parts of the body cannot be insisted upon too strongly. The connection between the eye and a viscus situated in the abdomen is just as close through the nervous, circulatory, and lymphatic systems, as that between any other two separate organs, and we find that the eye reacts to troubles located in distant parts just as other organs react to troubles in the eye. Not infrequently we have nausea and vomiting occasioned by ocular disturbances, and sometimes we have all of the symptoms of asthenopia and eyestrain induced by gastric derangement. A very proper objection can be raised to calling these symptoms by the name of eyestrain when their origin is not situated in the eye, but as in all cases they are the product of a nervous irritability induced by a fault in some organ or other, so that they are in reality reflex, and as there is nothing about them by which we can determine in what organ the trouble is situated, it is convenient to consider them all under one name. We can then speak of **nervous** asthenopia or eyestrain when the causative condition is situated in the nervous system, and of **reflex** asthenopia or eyestrain when the symptoms are excited by trouble outside of both the nervous system and the eye. The last could be divided, perhaps, into as many varieties as there are organs in the body, morbid conditions of the blood, or toxic causes competent to produce it, but this would give us an innumerable number of forms that differ only in the location of the fault. Nevertheless it is a common custom to speak of these symptoms when they are excited by abnormal conditions in the nose as **nasal** asthenopia or eyestrain, because a great

deal of attention has been called to this variety of late, and it may be that as more regard comes to be paid to the other varieties we shall become accustomed to speak of uterine, gastric, and other forms, or that a better term will be devised for these reflex symptoms, no matter what their origin. At present we shall include all of the cases which present symptoms of eyestrain that are dependent on causes to be found outside of the eye under two headings: nervous asthenopia, which may be made to include not only organic lesions of the central nervous system, but also functional derangements that are due to toxic causes, or to impoverished conditions of the blood which do not seem to depend on organic lesions elsewhere, and nasal asthenopia, which may serve as a type of the eyestrain induced by organic troubles in other organs.

Nervous Asthenopia

First among this class of cases comes a very large group in which the patients are suffering from the results of over work, over worry, and lack of rest. When an energetic hard worker tells us that he has to drive himself to do the work which he has always been accustomed to enjoy, that he dreads it and has to spur himself on to perform his daily duties, or, if he will not admit the loss of enjoyment, acknowledges that he feels the pressure of a sense of duty much more than he did, we have to consider whether he is not threatened with a nervous breakdown, and whether the symptoms of eyestrain which he presents are not produced by the **general nervous irritation.** In such cases the symptoms are of fairly recent date, and have been steadily progressive, so that, as a rule, we have little difficulty in recognizing nervous exhaustion to be the probable cause, except when the patient is aware of his own condition, is determined to conceal it, and refuses to admit anything that might indicate his need of the rest he proposes to avoid.

In a case in which the history and the onset of symptoms indicate a **nervous exhaustion** of this nature it is not always wise to attempt to relieve the symptoms of eyestrain at once, even though we may find a refractive error, or a muscular imbalance, the correction of which ameliorates the symptoms, for it often happens that the relief given by the correction impels the patient to persist in his work until he is completely broken down. An example of this nature was furnished by a gentleman sent by a physician, who was trying

to get him to let up in his work, to have his eyes examined in search for the cause of his sufferings. The correction of a slight refractive error so overjoyed the patient by a relief of his symptoms that, in spite of the urgent warnings on the part of both his physician and myself, instead of lessening he increased his work, and became totally prostrated a few months later. Such cases are not uncommon, and as it is human nature to neglect warnings unless they are backed up by tangible symptoms, we should eliminate nervous exhaustion if possible lest we do harm by a correction that seems to be indicated at the time, but is not the fundamental cause. The tests for heterophoria are particularly apt to be unreliable and misleading, because when one is very tired a slight incoordination may well be expected to appear in the muscles of the eyes, as it is known to do in other muscles of the body. After the nervous system has had a chance to recuperate from its exhaustion through a suitably long vacation from work, or through the removal of the causes for worry, we may find that the muscular faults have disappeared, and that the refractive errors were not the source of the trouble. A case in point was that of a man 38 years old, who was over worked, presented symptoms of eyestrain, and gave a history of a former attack of nervous prostration. He had a manifest hypermetropia of half a diopter, one degree of exophoria, and no hyperphoria, according to the phorometer, although the sursumvergence of his eyes differed by one degree. During a vacation of five months his symptoms gradually improved, and when he returned at the end of that time they were gone, he was hungry for work, as he expressed it, the acuteness of his vision had greatly improved, he could no longer see at a distance with his glasses, and his muscular balance was perfect. So far as his eyes were concerned he needed only the correction of his hypermetropia for reading, and during the past ten years the only attention they have needed has been due to the onset of presbyopia. All symptoms of eyestrain were done away with completely and permanently by the vacation, followed by a persevering avoidance of over work.

Symptoms of asthenopia, or of eyestrain, sometimes are met with in anæmia, and in convalescents who are debilitated, when perhaps they are caused by the **impoverished condition of the blood.** In these cases the correction of refractive errors is not apt to give relief, while the heterophoria is likely to be variable and puzzling. The same symptoms occur in neurasthenia, and in hysteria, in which

the heterophoria, some form of which commonly is present, seems to be ***symptomatic*** rather than causative, as a rule. Some writers have thought that these symptoms may be caused by a condition of anæsthesia, or of hyperæsthesia of the retina, but if any such cases have come my way they have not been diagnosed correctly.

In the great majority of cases in which asthenopia or eyestrain occurs in connection with a grave **disease of the central nervous system** it develops independently from some other cause. In a minority the symptoms induced by ocular trouble may simulate those of nervous disease, as in cases of chorea and epilepsy that have been cured through the treatment of an existing heterophoria, but in rare instances they appear to have been caused by the disease itself. *Hansell* and *Reber* have reported a case in which asthenopia with hyperphoria seemed to have been the first symptom of the onset of tabes, with a reflex immobility of the pupil as the second. The hyperphoria passed away spontaneously within a year. We cannot make a diagnosis of this nature except after a prolonged observation of the case.

Little is known of the **toxic** causes of asthenopia, but we know that hot, burning, hyperæmic eyes, with more or less headache and nervousness, are apt to be seen after a night's debauch. An acquaintance of mine cannot smoke a single cigar without suffering the next day from red, burning eyes that incapacitate him to a certain extent for work. Commonly we attribute the asthenopia in such cases to the hyperæmia of the conjunctiva and of the margins of the lids, and the balance of the nervous symptoms to toxic effects, but there was no hyperæmia of the conjunctiva in a patient whose smarting eyes I tried in vain to cure until he was induced to stop smoking five or six cigarettes a day. At the end of a week of abstinence with no other treatment his eyes were well; after smoking as usual for a week they were as bad as ever, and abstinence thereafter resulted in a perfect cure. It seemed to be demonstrated in this case that the cause of the asthenopia was the toxic effect of tobacco upon the nervous system, and it hardly seems reasonable to exclude the other cases from the same explanation because of the added symptom of a vasomotor dilatation of the blood vessels of the conjunctiva.

Nasal Asthenopia

Pressure in the nose is able to give rise to symptoms not only of asthenopia, but to all of those indicative of eyestrain, and these symptoms may seem to be so clearly dependent on some ocular fault that it is almost impossible at times to convince not only the patient, but even to realize ourselves, that the trouble is not in the eyes. When the symptoms of eyestrain are not relieved by a correction of the refractive errors my experience has led me to examine the nose as the place where the cause is next most likely to be found. Personal impressions are absolutely untrustworthy for a variety of reasons, so it may be wrong to consider nasal troubles to be a more frequent cause of eyestrain than heterophoria, but there are no statistics dealing with the relative frequency of nasal asthenopia and of heterophoria from which such an impression can be corrected.

The first writer of whom I am aware to call attention to the nose as the possible cause of asthenopia was *Gruening* in 1886, and perhaps the second was myself in 1894, but the subject did not attract much attention until during quite recent years. We know now that in many cases the pressure in the nose is at a point where it causes no obstruction to the breathing, and that in such cases the patient may have very good reasons for believing that he has never had any trouble in that organ, as the pain is referred to the eye and head, and seldom if ever is local. The most active nasal conditions in this class of cases are pressure contact and hyperæsthesia of the mucous membrane, of which the following discussion is taken from *Ziegler:*

"The first and most important causative agent is a condition which we may term pressure contact. The middle turbinate is usually the offending member, and is so often wedged in between the two vascular cushions of an engorged inferior turbinate and a sensitive septal puff that many mystifying reflex impulses are originated, the most pronounced of which are localized muscular twitchings, or choreiform movements of the face, head, and neck, while intense frontal headache and eyeache are more frequent effects. The middle turbinate, however, is not always the offending member. I have seen an inferior turbinate cause similar disturbances which were promptly relieved by the removal of the spur. The pull of contracting adhesions between the middle turbinate and the septum may also cause annoying reflexes. Another important ætiological ele-

ment is hyperæsthesia of certain areas in the upper air chambers of the nose. These sensitive points are most frequently located in an area covering the tubercle of the septum, which when irritated, quickly becomes engorged with blood and thus makes pressure against the closely approximated middle turbinate. Irritation of this sensitive area is so provocative of distinctly localized eye symptoms that I have long since dubbed it **'the eyespot of the nose.'"**

Another element that needs to be taken into consideration is that of nasal obstruction, but this is much more likely to attract the attention of both the physician and the patient, while it may perhaps be of less importance as the cause of eyestrain because it is apt to act indirectly by increasing the sensitiveness of the whole system and so rendering it more liable to reflex disturbances. Inflammation in the accessory sinuses also may act indirectly in this manner, but it acts directly as well through the pressure exerted upon the walls of the cavities by the accumulated secretion.

In a great many of the cases of nasal asthenopia the symptoms come on, or are aggravated, during reading, writing, or other near work, and a useful test is to have the patient take his **accustomed position** for reading and maintain it for a few minutes with his ***eyes closed.*** If the symptoms come on just the same as when he is actually reading or writing, the cause can hardly be in the eyes, but it probably is located in the nose, because the usual position for near work is apt to compress the blood vessels in the neck enough to induce an over supply of blood to the nose and so to increase any pressure contact that may be there.

Another diagnostic test which is very useful is to **press** with a bit of wet cotton on an applicator against the point where the middle turbinate seems to touch the septum. If our eyes have deceived us and there is no contact, the probe will pass between these tissues and cause nothing but local irritation. If the contact is real the probe will be arrested, and if its pressure causes the headache to increase at once, together perhaps with pain and weeping of the eye, we have reason to feel confident that we have discovered the source of the eyestrain, while if nothing except local irritation is produced the cause probably is elsewhere. As *Ziegler* has pointed out, the middle turbinate frequently is so hidden by the swelling of the other tissues in the nose that we are obliged to shrink the inferior turbinate before we can get a sight of the place which is the commonest seat of trouble. This **shrinking of the tissues** may be accomplished with

either adrenaline or cocaine carefully applied with cotton on an applicator, and forms of itself another valuable test. Occasionally it brings into view an unsuspected spur of the septum that impinges on one of the turbinates, and sometimes this can be demonstrated to be the cause of the symptoms by their instant cessation as soon as the apex of the spur is freed from the tissues into which it is pressing. Similarly the immediate amelioration of the symptoms as soon as a point of pressure, where there is no spur, is relieved gives us pretty definite information concerning the nature and location of the trouble. It is seldom possible to ascertain the presence or absence of pressure by inspection alone.

This shrinking of the tissues may reveal an inflammation of the frontal sinus, or of the anterior ethmoidal cells, as the cause of the symptoms, particularly when a gradual rather than a sudden alleviation of them is accompanied by an increase of clear fluid in the nose. In such a case we make a diagnosis of **catarrhal sinusitis** and understand that the escape of the secretion has been obstructed. If pus appears in the nose the inflammation is purulent and the patient has an **empyema** of the sinus. It is also possible for the engorged and hypersensitive condition of the mucous membrane about the eyespot of the nose to be due to such a sinusitis. In such a case a great amount of relief is apt to be experienced suddenly from the shrinking of the tissues, followed by a gradual amelioration of such symptoms as remain, while an increase of fluid, or perhaps of pus, appears in the nose. Then our diagnosis is again of sinusitis, rather than simply of intranasal pressure.

The importance of, as well as the difficulties met with in making the diagnosis of nasal asthenopia may be illustrated by a few cases which are reported in more detail in the Annals of Ophthalmology for October, 1909.

The first case has been under observation since 1893, was reported first in 1894, and is given place here because it is seldom that such a typical case can be watched for so long a time. The patient was a seamstress, 31 years old. She complained of headache and of severe pain in both of her eyes, but especially in the right, which was constant, but greatly aggravated whenever she made any attempt to sew or to read. She had simple hypermetropic astigmatism in both eyes, which was corrected without giving any benefit. Tests of the muscles showed orthophoria with a subnormal adduction. Pressure was discovered between an eburnated right middle tur-

binate and the septum, that required several operations to remove, in spite of the facts that she had never been aware of any nasal trouble, and that there was no obstruction to the breathing. During these operations she felt acute pain in her right eye, but none in her nose. The relief to the symptoms of eyestrain was complete, and she had no further trouble with her eyes until the onset of presbyopia. In 1906 a hyperphoria developed which was relieved by a prism. In 1912, twenty years after the operations had been performed on her nose, the old symptoms of eyestrain reappeared, and were relieved immediately by the division of a synechia which had formed between the remains of the middle turbinate and the septum. With the exception of these incidents and the onset of presbyopia, this patient has had no signs of trouble with her eyes in twenty-four years.

A lady 44 years of age complained of nausea, headache, and asthenopia when reading. An uncorrected compound hypermetropic astigmatism seemed to furnish an ample explanation, but its correction had no effect. A note to her physician, who was accustomed to intranasal work, called attention to a very slight, firm point of pressure between her right middle turbinate and a spur of the septum, but he was not able to find it, which was not very surprising as the nose otherwise was perfectly healthy. Finally the removal of this point of pressure completely relieved the symptoms.

A lady 45 years old had pain and "dizziness" in her eyes whenever she used them for near work, and obtained no benefit from the correction of a slight amount of hypermetropia and presbyopia. She knew that her nose was all right, but bony contact was found between the right middle turbinate and the septum. Removal of this eliminated her trouble.

A business man 34 years old had headaches and blurred vision, which were relieved for a few months by the correction of a low degree of compound hypermetropic astigmatism, but then recurred and became as bad as ever. The correction of a degree of hyperphoria with a prism was of benefit for only a few days. He assured me that he had never had any trouble in his nose aside from " a cold now and then, just the same as everybody else," yet examination revealed a large deviation of the septum with a thick vertical ridge impinging on the right middle turbinate. Other abnormalities were present to such an extent that a rhinologist in consultation advised an extensive operative intervention, which was declined. Finally he

consented to have me saw away the ridge, and this operation gave him positive and complete relief. A curious fact was that a year later his astigmatism had decreased one half, and two years later had disappeared. When last heard from he had no headache or other symptoms of eyestrain, although he was using his eyes very hard.

A very puzzling case was presented by a lady who had been under the care of a specialist in a neighboring city, had been wearing prismatic glasses, and had been sent to a very prominent specialist in New York City who found her muscular imbalance extreme. She had a compound hypermetropic astigmatism at an oblique axis in one eye alone, which beyond doubt was responsible for a part of her symptoms. She had worn prisms for some time, and without the proper correction of her refractive error she had a considerable degree of hyperexophoria, but with the correction on the hyperphoria disappeared, there was orthophoria by the screen test, to which she was not accustomed, and the exophoria as shown by the phorometer was reduced one half after the glasses had been worn for two days. After that time every attempt to add a prism caused discomfort in the eyes. Examination of the nose revealed a slight deviation of the septum to the right, bony pressure between the right lower turbinate and the septum, and an adhesion between the right middle turbinate and the septum. The symptoms of eyestrain persisted until these points of pressure had been removed, and then it disappeared. Two years later she reported that she had had no return of her asthenopia or of her headaches.

After we have excluded the eye and the nose as the probable seat of the trouble we must consider the possibility of its situation in any other organ of the body. In women we sometimes find that symptoms of eyestrain accompany **menstrual disorders.** An attack of headache associated with pain in the eyes, with or without such other symptoms as photophobia, lacrimation, weariness, hyperæmia of the conjunctiva, or occasionally the formation of minute pustular lesions in the margin of the lid, which recurs month after month and year after year before each menstrual period, can scarcely be referred to any other cause. We meet with more constant symptoms sometimes in **diseases of the kidneys,** and of other organs. In several obscure and obstinate cases of mine the diagnosis has finally been made through a thorough investigation of the **intestinal tract** and the benefit that accrued to the eyestrain from

appropriate treatment directed to the condition found in that viscus. A point that may possibly prove of value in the differentiation of cases of eyestrain that are dependent on lesions in the abdominal organs is one to which reference has been made already, a **heterophoria that fluctuates** in intensity from day to day, or perhaps on different hours during the same day. In the gastric cases it is often difficult to decide at once whether the disturbance is a reflex one of the stomach caused by the eyes, or one of the eyes from the stomach, as the symptoms and the history are apt to be exactly the same in both cases. The importance of the differentiation is self evident, yet sometimes it can be made only from the results obtained by treatment. As ophthalmologists it is natural that we should correct the faults we find in the eyes first, and when these fail turn the patient over to some other physician for attention to the stomach, but we should not carry the treatment of the eye defects to an extreme until the possibility of gastric trouble has been excluded.

Simulation of eyestrain is met with very rarely, and can scarcely be detected as long as the simulator confines himself to complaints of pain in the eye and head, for we can never be absolutely certain that we have excluded every possible cause of a reflex pain. Most of these simulators are reporters who wish material for a sensational article in a newspaper. After the exclusion of these, and of the victims of neurasthenia and hysteria, the only case of this nature that has come under my observation was that of a young man who for some reason was determined to secure a prescription for glasses from some well known ophthalmologist. It speaks well for the general accuracy of observation among eye surgeons that he did not succeed in his first attempts, though he did finally through a persistent complaint of pain in the eyes and brow, after he had submitted to a prolonged course of atropine which revealed a slight error of refraction.

CHAPTER XX

Psychical Symptoms Associated with the Sense of Vision

Reference has been made already to several conditions that may be termed psychical, as no lesion can be found in the eye to explain their existence, such as scintillating scotoma, and some faults in the field. In addition to these a number of curious phenomena have been met with occasionally that appear to involve certain of the connections between the sense of vision and various centers in the brain, and consequently may be brought to our attention when the eyes themselves appear to be organically and functionally perfect. The subjective symptom is striking in each case, whether it is blindness, an inability to name objects that are seen, or visual hallucinations, and is of diagnostic value, but it is not necessarily related to any other in this group.

PSYCHICAL BLINDNESS

Occasionally a child or an adult comes to us who appears to be blind, although he presents no lesion in his eyes that can explain the loss of vision. In a child we may detect some scars on the corneæ that can be recognized as having been caused by ulcers, and we inquire into his history. If we learn that he has recovered recently from a very prolonged attack of sore eyes during which he kept his lids tightly closed, we make a diagnosis of psychical blindness, and expect it to pass away before long. We know that when little children have suffered for months from a phlyctenular keratitis which maintained a constant blepharospasm, they sometimes appear to be blind for a while after the disease is well and they have begun to go about with their eyes open. The reason is unknown, but is supposed to be that they have temporarily lost the power to appreciate the meaning of the images formed on their retinæ, so that they have to learn again to see, just as they did in infancy. The prognosis is good.

We rarely find blindness in an adult which is not congenital, cannot be accounted for by the presence of objective symptoms, and has

lasted a longer time than would be necessary for symptoms to appear in the optic nerve if it were produced by poisoning, or by a lesion in either the orbit or the brain. It is then commonly associated with anomalies in other senses, such as those of touch, pain, temperature, taste, hearing, or smell, that guide us to a diagnosis of hysteria, or, if such a cause is indicated by the history, to one of psychosis due to traumatism.

VISUAL HALLUCINATIONS

Hallucinations occur most commonly in delirium and in certain types of insanity, but they are seen occasionally by persons who are sane, and in such cases the eyes should be examined. *Swanzy* states that he met with a case in which hallucinations occurred in connection with glaucoma, so the tension should be taken, and the papilla examined for a glaucomatous excavation. After this disease has been excluded the field should be tested for homonymous hemianopsia, and if this is found we may conclude that some sort of a cerebral lesion is irritating the visual memory center. If this is absent it is possible that we may find the hallucinations to have a homonymously hemianopic character, and it is said that such have been known to persist for years. We must bear in mind also that hallucinations may be one of the prodromal symptoms of epilepsy, and that they may appear after an attack of this disease.

CONDITIONS IN WHICH VISUAL CONCEPTION SEEMS TO BE DISSOCIATED FROM OTHER MENTAL FACULTIES

The conditions about to be described are far more apt to come under the observation of the neurologist than of the ophthalmologist, the number of reported cases does not appear to be large, and the author can lay no claim to personal experience with them. He has gleaned the descriptions from such neurological textbooks as were available, with the few references he has found made to them in works on ophthalmology. In nearly every case they are focal symptoms of lesions in the brain that interfere with the connections between the center for vision and such other centers as those for memory, speech, and color. The accuracy with which they guide us to the site of the lesion depends to a great degree on the exactness of our knowledge of the location of these centers.

Visual Aphasia

A patient may be able to see the various objects about him, and to know their uses, but be unable to call them by name, unless he can become cognisant of them through some other sense. He can see a spoon, a bell, or a lump of sugar, and be able to tell the uses of each, but cannot name any of them as long as he relies on his sight alone, and yet can name each as soon as he feels the one, hears the second, or tastes the third. Very few cases of this **visual** or **optical aphasia** have been reported, and some neurological writers seem to doubt the existence of one which was not associated with some other form of sensory aphasia. Right homonymous hemianopsia appears to have been present in every one. Some of the patients had alexia, others had not. According to *Bing* the cerebral lesion is supposed to be so situated as to interrupt the connection between the centers for the recognition of objects in the occipital lobe and Wernicke's sound memory center in the temporal lobe.

Visual Amnesia

Another patient is unable not only to name an object, but also to tell the use of it when he relies on sight alone, although he can do both when it appeals to one of his other senses. This is called by different writers **visual amnesia, optical agnosia,** and **mind blindness.** Sometimes the patient can describe the object from memory, although he cannot recognize it when seen, at other times he has lost all memory of it, even though he can describe the appearance of the object he sees. In the latter case the lesion is supposed to lie in the center for memory, in the former to be situated between that center and the one for vision.

Alexia

When a patient can name and comprehend the uses of the various objects about him, but finds written or printed letters or words to be meaningless symbols, we say that he has alexia, or **word blindness,** a condition which can appear more or less independently of other aphasias. If he can express his ideas in writing, or can write from dictation, although he cannot read what he has written, or copy written or printed words, he has subcortical, or pure alexia. When he cannot write at all, the other conditions remaining the

same, he has cortical alexia. A rare form that is said to have been met with is an alexia for the written notes of music, perhaps associated with an inability to understand music at all. Patients with alexia commonly present other cerebral symptoms, among which may be hemianopsia. Cortical alexia locates the lesion in the center for the visual memory for words, which most writers seem to think is situated in the left angular gyrus. *Swanzy* quotes *Dejerine* and *Wernicke* to the effect that in right handed people this center is in the left angular gyrus and the inferior parietal lobule. A subcortical alexia indicates that the lesion is so placed as to interrupt the connection between this center and that for vision.

Amnesic Color Blindness

When we meet with a patient who can match colors, but has forgotten their names, we shall probably find that he has a right homonymous hemianopsia, and say that he has amnesic color blindness, **color aphasia,** or **color amnesia.** The lesion probably will be found in such a place in the occipital lobe that it interrupts the path between the centers of color vision and of speech.

Dyslexia

A very few cases are on record in which a patient was able to see and to recognize words perfectly, but could read only a few before he was seized with so strong a feeling of aversion that he could not continue. He might return to the task in a moment only to turn away again with the same feeling of disgust. This dyslexia was sometimes the first symptom excited by a cerebral disease, but was soon followed by others. It seems to have been temporary, and to have recurred in some of the cases. When an autopsy was obtained the lesion was usually found in the third frontal convolution.

INDEX

Abduction, 104
Ablepharia, 15
Abnormalities of the fundus, 324
Abnormal tension of the eyeball, 279
Abrasions of the cornea, 123, 162, **163, 184**
Abscess, alveolar, 27, 222
  of the brain, 22, 338, 340
  of the cornea, 190
  of the lacrimal gland, 74
  of the lacrimal sac, 54
  of the lid, 27, 37
  of the orbit, 56
  of the vitreous, 275, **307**, 397
  of the zygomatic fossa, 61, 74, **75**
Absence, of a muscle, 94, 96
  of canaliculi, 53
  of puncta, 53
Absolute glaucoma, 195, 281, **291**
Absolute immobility of the pupil, 208, **244**
Accessory sinuses of the nose, 27, 29, 59, 60, 73, 74, 75, 77, 94, 120, 338, 431
Accommodation, paralysis of the, 296
  reaction of the pupils to, 206
  spasm of the, 296, **451**, 452
  weakness of the, 309, 450
Acne, 32
Acquired amblyopia, 410
Acquired color blindness, 424
Acquired conus, 359
Acquired cryptophthalmos, 40
Acquired nystagmus, 107
Acquired ptosis, 17
Acquired sclerosis of the choroid, 357
Acromegaly, 70, 332
Actinomycosis, 149
Acute catarrhal conjunctivitis, 27, 119, 122, 123, **124**
Acute infectious diseases, 20, 74, 127, 337, 372, 375, 379, 393, 394
  iritis in, 225
Acute inflammatory glaucoma, 217, 218, **282**, 439
Acute plastic iridocyclitis, 119, 215, 218, 261, 283
Acute purulent dacryocystitis, 27, 54, 73, 119
Addison's disease, 31, 194
Adduction, 104
Adenoma, of conjunctival glands, 37
  of the Meibomian glands, 36, 37
Adenopathy of lymphatic glands, 58
Adherent leucoma, 169, 236
Adjustment of glasses, 444
Adrenaline, toxic amblyopia from, 414
Ætiology of iritis, 219
Affections of the color sense, 420
Agnosia, optical, 469
Albinism, skin of the lids in, 31
Albinotic fundus, 322
Albuminuric choked disk, 339
Albuminuric retinitis, 374
  of pregnancy, **375**, 393
Alcohol and tobacco, toxic amblyopia from, 328, 411, 413, **415**
Alcohol, methyl or wood, poisoning, 330, 332, **415**
Alcoholism, 20, 108, 116, 207, 239, 245, 276, 439
Alexia, 435, **469**
  cortical, 470
  for music, 470
  subcortical, 469
Alopecia, 45
Alternating mydriasis, 242
Alveolar abscess, 222
Amaurosis, 381, **408**
  partial fugacious, 432
Amaurotic cat's eye, 395
Amaurotic family idiocy, 363, **383**
Amaurotic immobility of the pupil, 243
Amblyopia, 408, **409**, 418
  a cause of convergent strabismus, 83
  acquired, 410
  congenital, 348, **409**
  from bright light, 410
  from disease, 411
  from overwork, 416
  hysterical, 412
  reflex, 411
  toxic, 413
  toxic, from alcohol and tobacco, 328, 411, 413, **415**
  toxic, from quinine, 330, 332, 345, 413, **414**
  toxic, from wood alcohol, 330, 332, **415**
  traumatic, 410
Amnesia, color, 435, **470**
  optical, 469
  visual, 469
Amnesic color blindness, 424, **470**
Amyloid degeneration, **153**, 168
Anæmia, 52, 119, 217, 222, 276, 345, 348, 352, 378, 393
  asthenopia and eyestrain from, 120, 459
  iritis from, 234
  of the conjunctiva, 120
  of the fundus, 344
  pernicious, 345, 352, 378, 379
Anæmic retinitis, 378
Anæsthesia dolorosa, 180
Anæsthesia of the cornea, **158**, 179, 183, 186, 341
Anatomical faults a cause of convergent strabismus, 84
Angioid streaks in the retina, 353

Angioma, of the choroid, 405
of the conjunctiva, 115
of the lid, 31, 38, 39
cavernous, of the lid, 38, 39
of the orbit, 72
Angiomegaly of the lids, 31
Angiosclerosis, 354
Angle γ and strabismus, 87
Aniline oil, toxic amblyopia from, 413
Aniridia. (See Irideremia)
Anisocoria, 205, **240**
an early symptom of tuberculosis, 240
in diseases of the central nervous system, 241
Ankyloblepharon, 40, 151
Annular keratitis, 186
Annular scleritis, 196
Annular scotoma, 430
Anomalies, motor, without deviation, 101
Anomaly of deviation, congenital, 93
Anterior chamber, 198
deep, 200
obliteration of the, 199
shallow, 199
Anterior choroiditis, 389
Anterior ethmoidal cells, 59, 75, 463
Anterior lenticonus, 251
Anterior polar cataract, 169, **256**, 259, **260**
Anterior segment of the eye, infection of the, 306
Anterior staphyloma, 178, 195
Anterior synechiæ, 169
Anteversion of the iris, **211**
Anthrax, 28, 32, 33, 35, 58, 74
Aphakia, 211
Aphasia, color, 470
optical, 469
visual, 469
Apical infections of the teeth, 222
Aplasia of a nucleus, 94, 96
Apoplexy, 118, 239
of the retina, 379
Aqueous, visible changes in the, 200
Arc, light reflex, 206
Arcus senilis, 167
Areolar choroiditis, 389
Argyll-Robertson pupil, 245
Aridosiliquose cataract, **257**, 271
Arsenical poisoning, 45
Arterial pulse, 321
Arteriosclerosis, 117, 197, 236, 276, 331, 339, 340, 348, 354, 355, 366, 375
Artery, central, occlusion of the, 332, 336, 345, 363, **368**, 383
central, occlusion of a branch of the, 370
persistent hyaloid, 255, 259, **272**, **327**
Aseptic purulent iritis, 235
Aspergillus fumigatus in corneal ulcers, 174, 177
Asthenopia, 440
from anæmia, 459
from disease of the central nervous system, 460
from nervous exhaustion, 458
nasal, 452, 457, **461**
nervous, 457, **458**
reflex, 457
toxic, 460
Astigmatism, 156, **453**
against the rule, 156
Astigmatism, irregular, **141**, **156**
regular, 156
with the rule, 156
Atavistic defects, 13
Ataxia, hereditary, 108
Atrophic cup or excavation, 318
Atrophy, gyrate of the choroid and retina, 382
hereditary optic, 332
of the eyeball, 62, **292**
of the iris, 227, **236**, 245
of the skin of the lid, 31
neuritic optic, **329**, 337
optic, 182, **327**, 416, **421**
retinitic optic, 332
simple optic, 329
Attention reflex of pupils, 207
Atypical colobomata, 214
Aural nystagmus, 108
Autointoxication, 173, 190, 222, 393

Babies' sore eyes, 130
Background of the fundus, 322
Bartel's test for paralysis of the ocular muscles, 78
Basedow's disease. (See Exophthalmic goiter)
Beaded appearance of the retinal vessels, 355
Bee sting of the cornea, 260
Benign follicular affections of the conjunctiva, 141
Berlin's opacity. (See Traumatic œdema of the retina)
Binocular single vision, 84
in relation to convergent strabismus, 84, 85
Birthmarks, 31
Bisulphide of carbon, toxic amblyopia from, 329, 413
Black cataract, 271
luxated into the anterior chamber, 160
Black spots in the fundus, **346**, **381**, **387**
Blastomycetic dermatitis, 33
Blepharitis marginalis, 46
Blepharitis ulcerosa, 48
Blepharochalasis, 27, **31**
Blepharophimosis, 40
Blepharospasm, **24**, **467**
clonic, 24
hysterical, 25
occupational, 25
senile, 26
tonic, 24
Blindness, amnesic color, **424**, **470**
color, 420, 422
mind, 469
psychical, 467
word, 469
Blind spot, 431
Blood cyst, 69
Blood stain of the cornea, 159
Blood vessels of the retina, **319**, **350**
Boil on the lid, 37
Brain lesions, 19, 22, 95, 99, 180, 238, 239, 246, 338, 340, 375, 434, 439, 468, 469, 470
Brain, lesions at the base of the, 95, 180
Branch of central artery, occlusion of a, 370
Brawny scleritis, 196
Bridge coloboma of the iris, 210, **214**

Bulbar paralysis, 21, 97, 98
Bulla, 181, 183
Bullous keratitis, **181, 183**
Bundle of nerve fibers, disease of the papillomacular, **328**, 376, 431
Buphthalmos. (See Infantile glaucoma)
Burns, lime, 154, 163
  of the conjunctiva, 44, 117, 127, 146, **154**
  of the cornea, 154, **163**
Bursa of the trochlea, distended, 69

Cachectic cataract, 263
Cachexiæ, 31, 378
Calcareous cataract, 252, **257**
Calcareous degeneration of the cornea, 168
Calcification of the conjunctiva, 153
Calmette's test, 189, 230
Canaliculus, closure of the, 53
  mucocele of the, 53
Cannabis indica, toxic amblyopia from, 329, 413
Caput medusæ, 291
Carbon bisulphide, toxic amblyopia from, 329, 413
Carbon dioxide poisoning, 20, 97, 348
Carbuncle of the lid, 38
Carcinoma, of the choroid, 402, **406**
  of the lid, 39
  of the orbit, 70
Caruncle, 110, **111**
Cataphoria, 101
Cataract, 230, **252**
  anterior polar, 169, **256**, 259, 260
  arilosiliquose, **257**, 271
  black, 271
  blue punctate, 270
  cachectic, 263
  calcareous, 252, **257**
  caused by electric flashes and by electric shock, 258, 261
  caused by X-rays, 258, 261
  central, 254
  complicated, 227, 252, **262**
  congenital, **253**, 255
  cystic, 257
  degenerated, 252, **257**, 271
  diabetic, 259, **263**
  due to disease, 261
  false, 262, 264
  fluid, 252
  fusiform, 255
  glassblowers', 258, **261**
  glaucomatous, 259, **262**
  lamellar, 253
  luxation into the anterior chamber, 160
  membranous, 254, 257
  milky, 257, 258
  Morgagnian, 257, **271**
  nuclear, 269
  posterior polar, **255**, 259, 383
  punctate, **256**, 270
  pyramidal, 256
  senile, 252, 262, **264**
  senile, hypermature, 257, 265, **271**
  senile, immature, 265, **268**
  senile, incipient, 256, 265, **267**, 275
  senile, mature, 265, **268**
  senile, subcapsular cortical, 267
Cataract, senile, subjective symptoms of, 265
  siliquose, 254, **257**, 258, 271
  spindle shaped, 255
  supranuclear, 269
  total, **254**, 259
  traumatic, **257**, 262, 297
  zonular, **253**, 259
Catarrhal dacryocystitis, 53, 54
Catarrh, vernal, 134
  vernal, bulbar, 135
  vernal, mixed, 135
  vernal, tarsal, 134
Causes, of convergent strabismus, 82
  of divergent strabismus, 85
  of iritis, 220
  of secondary glaucoma, 289
  of vertical strabismus, 82
Cavernous angioma of the lid, 38
Cavernous sinus, thrombosis of the, 73, 76, 343
Cellulitis of the orbit, 27, 37, 56, 60, **73**, 119, 283, 339, 380
Central artery, anomaly of the, 273
  occlusion of the, 332, 336, 345, 363, **368**, 383
  occlusion of a branch of the, 370
Central cataract, 254
Central choroiditis, 243, 269, 363, 364, 372, 373, 385, 389, **390**
Central nervous system, diseases of the, 21, 209, 241, 243, 244, 246, 429, 430
Central retinitis, 366, 370, **372**, 385, **390**
Central scotoma, 298, 328, 339, 367, 376, **390**, **430, 431**
Central vein, thrombosis of the, 379
Centrifugal neuron, **206**, 208
Centripetal neuron, 206
Cerebellar disease, 100, 108, 180
Cerebral syphilis, 241, 244, 245, 246, **331**
Cerebrospinal meningitis, 20, 394
Chalazion, 25, **36**, 116, 122, 146, **152**
  giant, 37
Chancre, of the conjunctiva, 58, **151**
  of the lids, 22, **33**, 35, 36, 58
Chancroid of the lids, 34
Charcot's tabetic mask, 159
Chemosis, **118**, 183
  caused by drugs, **118, 119**
  filtration, 119
  fugacious, 119
Cherry red spot, **368**, 383
Chicken pox, 29, 32, 98
Chlorate of potassium, toxic amblyopia from, 414
Chloroform, effect on the pupils of, 239
Chloroma, or chlorosarcoma, 57, 71
Chlorosis, 119, 263, 276, 345, 348, 352, **378**, 379, 393
Choked disk, 329, 333, 335, **336**, 345, 357, 431
  a late symptom in tumors of the brain, 341
  albuminuric, 339
  atypical, 341
  diagnostic value of, 339
  differentiation from optic neuritis, 335
  early in the course of a tumor of the brain, 341
  early stage of a, 337

Choked disk, height of a, **336**
in intracranial disease exclusive of tumors, 343
in otitis media, 343
with tumors of the brain, 340
of both eyes, 340
of only one eye, 339
vision in, **335**, 343
Cholera, 116, 192, 345
infantum, 116, 192
Chorea, 241, 460
Chorioretinitis, **371**
Choroid and retina, 344
gyrate atrophy of the, 382
Choroid, angioma of the, 405
carcinoma of the, **402**, **406**
coloboma of the, 214, 326, 346, 357, 358, **361**, 387
conglomerate and solitary tubercle in the, 394, 396, 397
detachment of the, 199, **404**
endothelioma of the, 406
flat sarcoma of the, 406
gumma of the, 405
miliary tubercles in the, 392, **393**
poor, 323
rupture of the, 299, **364**, 387
sarcoma of the, 394, 397, 399, **405**
sclerosis of the, 346, **357**, 358, 361, **363**, 364, 385
tiger, 322
tuberculoma of the, 394, 399, 405
tuberculosis of the, 393
tumor of the, differentiated from detachment of the retina, 402
tumors of the, 394, **405**
vessels of the, 322
Choroidal hemorrhages, **346**, 405
Choroiditis, 182, 217, 227, 255, 259, 262, 332, 346, 357, **381**, **383**, **385**, **386**, 388, 432
anterior, 389
areolar, 389
central, 243, 269, 363, 364, 372, 373, 385, 389, **390**
diffuse, 389
disseminated, 338, 389, **391**
early stage of, 388
hereditary, 392
metastatic, 374
myopic, 263, 391, 392
purulent, **394**, 397
Tay's, 373, 390
tuberculous, 393
varieties of, 389
Chromatopsia, 420
Chromidrosis, 32
Chronic conjunctivitis, 121
an occupational disease, 122
Chronic inflammatory glaucoma, **284**, 439
Chronic progressive ophthalmoplegia, 16, 21, **98**
Chronic purulent dacryocystitis, 54
Cicatricial adhesions a cause of strabismus, 82
Cicatricial ectropion, 41
Cicatricial entropion, 44
Cicatrix in the cornea, **161**, **166**, 168
Ciliary body, wounds of the, 308
Ciliary muscle, spasm of the, 296, **451**, **452**
tone of the, 448, 449, 453
Ciliary muscle, weakness of the, 309, 450
Ciliary staphyloma, 195
Ciliary zone, 202
Cilioretinal vessels, **321**, 369
Circinate degeneration of the retina, **363**, **386**
Circinate retinitis, 386
Climacteric, 190, 231
Clinoscope, 105
Closure of the lids, reaction of the pupils to the, 207
Cocaine, toxic amblyopia from, 414
Coffee, toxic amblyopia from, 415
Colloid formations on the vitreous lamella of the choroid, **349**, 374, 386, 390
Coloboma of the choroid, 214, 326, 346, 357, 358, **361**, 387
defect produced in the visual field, 362
rudimentary, 361
Coloboma of the iris, **213**, 250, **361**
artificial, 214
atypical, 214
bridge, 210, **214**
false, 214
partial, 213
total, 213
Coloboma of the lens, 251
Coloboma of the lid, 15
Coloboma of the macula, 346, 357, 358, **363**, 387
Coloboma of the optic nerve sheath, **318**, **326**, 358
Colocynth, toxic amblyopia from, 413
Color amnesia, 435, **470**
Color aphasia, 470
Color blindness, 420, **422**
acquired, 424
amnesic, 424, **470**
congenital, 423
detection of, 424
Color, fields for, 412, 427, **428**, 429
of the iris, 203
of the lashes, 45
of the papilla, 315
of the retinal vessels, 352
scotoma, 411, 415, 426, 427
sense, affections of the, 420
vision, 421
Coma, 178, 291
Complicated cataract, 227, 252, **262**
Concentric contraction of the field of vision, 286, 381, 403, 419, **429**
Conditions in which visual conception seems to be dissociated from other mental faculties, 468
Congenital amblyopia, 348, **409**
Congenital anomaly of deviation, 93
Congenital cataract, **253**, 255
Congenital color blindness, 423
Congenital conus, 359
Congenital cryptophthalmos, 40
Congenital defects of the conjunctiva, 112
Congenital defects of the lids, 15
Congenital ectopia of the lens, 250
Congenital ectropion, 43
Congenital exophthalmos, 64
Congenital irideremia, 212, 250
Congenital nystagmus, 107
Congenital ptosis, 16
Congenital opacities of the cornea, 162

Congenital sclerosis of the choroid, 357
Congestion of conjunctival vessels, 120, 124
Congestion of episcleral vessels, 120, 124
Conglomerate tubercle of the choroid, 394, 397
Conical cornea, 157
Conjugate paresis, 92, **99**
  hysterical, 100
Conjunctiva, 110
  amyloid degeneration of the, 152
  anæmia of the, 120
  benign follicular affections of the, 141
  burns of the, 154
  calcification of the, 153
  chancre of the, 58, **151**
  concretions in the, 122
  congenital defects of the, 112
  cysts of the, 115
  emphysema of the, 118
  foreign body on the, 120, 121, **122**
  gumma of the, 152
  hemorrhage beneath the, 117
  hyaline degeneration of the, 152
  hyperæmia of the, 121
  leprosy of the, 154
  lime deposits in the, 153
  lithiasis of the, 153
  mucous patches on the, 152
  pemphigus of the, 151
  simple herpes of the, 150
  spots on the, 113
  syphilis of the, 151
  tuberculosis of the, 28, 58, 128, 136, **145**, 152
  tumors of the, 114
  ulcers of the, 58, 146, 152
Conjunctival congestion, 120, 124
Conjunctival injection, 120, 124
Conjunctivitis, 58, 120
  acute catarrhal, 27, 119, 122, 123, **124**
  acute catarrhal, caused by traumatism, 127
  acute catarrhal, micro-organisms that cause, 125
  chronic, 121
  croupous, 132
  diphtheritic, 132, **133**, 172
  diplobacillus, 122
  follicles induced by, 142
  follicular, 144
  gonorrheal, 58, **128**, 134, 172
  gonorrheal, of adults, 128
  gonorrheal, of infants, **130**, 171
  membranous, 116, 117, **131**, 146
  metastatic gonorrheal, 129
  Parinaud's, 58, 146, **147**
  petrificans, 153
  phlyctenular, 29, 58, 136, **149**
  purulent, 116, 129
  squirrel plague, 127
Connective tissue ring, 316
Consensual reaction of the pupil to light, 205
Consequences of a perforation of a corneal ulcer, 168
Contraction furrows or folds of the iris, 202
Contraction of the field of vision, 286, 381, 403, 419, **428**
Contusions of the eye, 211, 237, 244, 258, 259, **294**, 371, 404
  secondary glaucoma after, 289, 299
Contusions of the eye, subnormal tension after, 299
  with rupture of the capsule, 300
  without rupture of the capsule, 294
Conus, 346, 357, **358**, 361, 387, 431
  inferior, 326, 357, 358, **361**, 387
Convergence, myotonic reaction of the pupils to, 207
  paralysis of, 92, 100
  reaction of the pupils to, 206
Convergent strabismus, 79, **82**
  causes of, 82
  hysterical, 82
  simulated, 82
Corectopia, **209**, 250
Cornea, 156
  abrasions of the, 123, 162, **163**, 184
  abscess of the, 190
  anæsthesia of the, **158**, 179, 183, **186**, 341
  arcus senilis of the, 167
  blood stain of the, 159
  burns of the, 163
  congenital opacities of the, 162
  conical, 157
  cysts of the, 171, 193
  degenerations of the, 161, **167**, **291**
  ectasia of the, 169
  febrile herpes of the, 181
  fistula of the, 169
  foreign body on the, **122**, 159, **162**
  hyperæsthesia of the, 158
  infiltrate of the, 161, **164**
  opacities of the, 161
  pigmentation of the, 159
  siderosis of the, 160
  staphyloma of the, 169, **170**, 193
  surface of the, in interstitial keratitis, 188
  tumors of the, 170, **192**
  ulcers of the, 162, **164**, 187, 215
  wounds of the, 162
Corneal cicatrix, 161, **166**, 168
Corneal degeneration, calcareous, 168
  Groenouw's nodular, 167
  ribbon shaped, 167
  Uhthoff's, 168
Corneal horn, 170
Corneal ulcers. (See Ulcers of the cornea)
Coronula, 368
Cortical reflex of the pupils, 207
Cover test, 103
Crater pupil, 198, 209, 227, 231, 261
Crescentic ulcer of the cornea, 165, 173
Cretinism, 31
Crick dots, 349
Croupous conjunctivitis, 132
Cryptophthalmos, 40, 112
Crypts of the iris, 202
Cup. (See Excavation of the papilla)
Cutaneous horns, 39
Cyanosis of the lids, 29
Cyclitis, 162, 191, 200, **216**, 225
  recurrent chronic, 217
Cyclophoria, 101, **105**
Cycloplegics, use for diagnosis, 125, **289**, 451, 452
Cystic cataract, 257
Cysticercus, 116, 237, 278
Cysts, hydatid, 57, 69
  of the conjunctiva, 115

Cysts, of the cornea, **171, 193**
of the iris, 201, 237
of the lacrimal gland, 57
of the Meibomian glands, 35, 37
of the orbit, 38, 57, 69
of the retina or vitreous, 363, 397, **405**
retention, 39, 57

Dacryoadenitis, 56
acute, 56, 74
chronic, 57
exophthalmos from, 74
Dacryocystitis, 27, **53, 175**
acute purulent, 27, 54, 73, **119**
catarrhal, 53, 54
chronic purulent, **54**
of infants, 54
Dacryops, 52, **57**
Danger zone of the sclera, **307**
Declination, 83, 106
of the vertical meridian of the eye a cause of convergent strabismus, 83
Deep anterior chamber, 200
Deep punctate keratitis, 191
Deep purulent ulcer of the cornea, 165
Defects, about the papilla and the macula, 358
in the field of vision, 362, 370, 408, **426**
in the field of vision, sector shaped, 409, **430**
in the lids, 15
Degenerated cataract, 252, **257, 271**
Degeneration, circinate, 363, **386**
macular, 373, 377, **383**
maculocerebral, 373, 377, **383**
of the conjunctiva, amyloid, 152
of the conjunctiva, hyaline, 152
of the cornea, 161, **167**, 291
of the cornea, Groenouw's nodular, 167
of the cornea, ribbon shaped, 167
of the cornea, Uhthoff's, 168
of the macula, senile, 269, 372, 373, 390, **391**
of the retina, pigmentary, 255, 259, 332, 350, **380**, 385, 431
Demodex folliculorum, 48
Dendritic keratitis, 166, **180**
Dental infection, 221, 222, 411
Dermatitis, blastomycetic, 33
herpetiformis, 30
Dermatolysis of the lids, 31
Dermoid, of the conjunctiva and cornea, 112, 114, 115, 193
of the lid, 38
of the orbit, 70
Descemet's membrane, deposits on, 191, 216, 230, 232
mutton fat deposits on, 191, 217
wrinkling of, 191
Detachment, of the choroid, 199, **404**
of the retina, 262, 299, 300, 396, **399**, 433
of the retina, bullous, 396, 401
of the retina, flat, 336, 401
of the retina, in children, 403
of the retina, prodromal symptoms of, 399
of the retina, without discoloration of the fundus, 402, 403
Detection of color blindness, 424
Detection of foreign bodies by the X-rays, 304
Deviation, congenital anomaly of, 93
primary, 80
secondary, 80
Deviations of the eyeball, 62, **78**
Diabetes, 98, 99, 118, 197, 207, 222, 234, 235, 276, 338, 339, 348, 366, 375, 376, 379, 391, 392, 393, 411, 432, 439
Diabetic cataract, 263
Diabetic retinitis, 376
Diagnosis, 1
Diagnostic use of cycloplegics and of mydriatics, 8, 125, 209, **218**, 233, 238, 267, 289, 451, 452
Diagnostic value of choked disk, 339
Diagnostic value of optic neuritis, 337
Differentiation, 13
of acute catarrhal conjunctivitis, 124
of a staphyloma of the cornea from a tumor, 170
of chancre of the conjunctiva, **151**
of choked disk from optic neuritis, 335
of conus from posterior staphyloma, 361
of detachment of the retina from tumor of the choroid, 402, 403
of episcleritis, 197
of eyestrain due to heterophoria, 455
of eyestrain due to refractive errors, 445
of follicular conjunctivitis from trachoma, 144
of glioma from pseudoglioma, 398
of hemorrhagic glaucoma from glaucoma with hemorrhages, 290
of lesions productive of muscular paresis, 94
of maculocerebral and macular degenerations of the retina, 385
of nasal asthenopia, 462
of the nature of a corneal opacity, 161
of Parinaud's conjunctivitis from tuberculosis of the conjunctiva, 146, 148
of phlyctenules, 150
of physiological from pathological excavations of the papilla, 318
of a pneumococcal from a diplobacillary ulcer of the cornea, 176
of retinitis, 373
of school folliculosis from trachoma, 143
of strabismus from muscular paresis, 79
of vernal catarrh, 135
Diffuse choroiditis, 389
Diffuse opacity of the media, 334
Dilatation of pupils, reflex, 208
Dinitrobenzene, toxic amblyopia from, 414
Diphtheria, 98, 132, 133, 289, 337, 394
Diphtheritic conjunctivitis, 132, **133**, 172
Diphtheritic ulcer of the lid, 32, 34
Diplobacillus, conjunctivitis caused by the, 122
ulcer of the cornea caused by the, 175, 176
Diplopia, in deviations of the eyeball, 81
monocular, 296
Direct method, **10**, 402
Direct reaction of the pupils to light, 205
Discolorations, of the lids, 29, **31**
of the lids by grains of powder or metal, **31**

Disease, amblyopia from, 411
of the papillomacular bundle of optic nerve fibers, **328**, 339, 431
Disklike keratitis, 186
Dislocation of the lens, 209, **247**
Displacement of the field of vision, 429
Displacements of the eyeball, 62
Disseminated choroiditis, 338, 389, **391**
Disseminated sclerosis, 99, 100
Distichiasis, 45
Divergence insufficiency, a cause of convergent strabismus, 84
Divergence, paralysis of, **93**, 100
Divergent strabismus, 79, 82, **85**, 341
causes of, 85
Dots, Gunn's, **349**, 373
metallic, 349
Drugs, follicles induced by, 142
Drusen, 349
Duane's table for locating a paretic muscle, 90
Dysentery, 116, 192
Dyslexia, 435, **470**

Early papules of the iris, 228
Ecchymoses of the lids and conjunctiva, **28**, 117, 150
Eclampsia, 347
Ectasia of the cornea, 169
Ectopia of the lens, 211, **249**, 257
Ectropion, **41**, 52, 178
cicatricial, 41
congenital, 43
of the uvea, 202
paralytic, 42
senile, 42
spastic, 42
Eczema, 29, 32, 49, 172
of the margin of the lid, 48
Eczematous keratoconjunctivitis, 172
Elephantiasis, 32
lymphangiectatica, 39
Emmetropia, 448
Emphysema, of the conjunctiva, 118
of the lids, 28
of the lungs, 276
of the orbit, 66
Empyema, of the anterior ethmoidal cells, 59, 75, 463
of the frontal sinus, 37, 59, 75, 463
of the maxillary sinus, 60, 75
of the posterior ethmoidal cells, 61, 431
of the sphenoidal sinus, 61, 431
Encephalocele, 38, 59, 69, 72
Endarteritis obliterans, 355, 369
Endophlebitis obliterans, 355, 380
Endothelioma, of the choroid, 406
of the lid, 35
Enophthalmos, 62
bilateral, 62
during movements of the eye, 63
in lesions of the sympathetic nerve, 63
in migraine, 63
slight unilateral, 62
traumatic, 63
Enteritis, 225, 235
Entropion, **43**, 52, 140, 141, 151
cicatricial, 44
congenital, 43
Entropion, senile, 43
spastic, 43
Epicanthus, 15
Epilepsy, 25, 238, 239, 241, 460, 468
Epiphora, 51
Episcleral congestion, 120, 124
fugitive, 61
Episcleral injection, 120, 124
Episcleritis, 150, **197**
periodica fugax, 198
Epithelioma, of the conjunctiva, 114, 136, 152
of the cornea, 193
of the lids, 32, **35**, 36
Equatorial staphyloma, 195
Ergot, toxic amblyopia from, 345, 414
Erosion, recurrent, 163, 184
Erroneous ideas concerning the wearing of glasses, 445
Errors of refraction, 121, 345, 443
varying effects produced by, 444
Eruptions on the lids, 29
Erysipelas, 28, 30, 32, 35, 77, 119, 337, 345
Erythropsia, 421
Esophoria, 84, 101
a cause of convergent strabismus, 84
Esotropia, 82
Ether, effect on the pupils of, 239
Ethmoiditis, 38, 59, 119
Eversion of the lids, 41, **111**
Examination of the inverted image, 9
Examination of the upright image, 10
Excavation of the papilla, atrophic, 318
glaucomatous, **281**, 318
physiological, **317**, 318
physiological and pathological, 318
Excluded pupil, 209, 227
Exophoria, 101
Exophthalmic goiter, 22, 31, 43, 45, 52, **65**, 207, 242
nuclear paresis in, 99
winking in, 23
Exophthalmos, **63**, 178, 288
caused by abscess in the zygomatic fossa, 74
caused by acromegaly, 70
caused by angioma of the orbit, 72
caused by cerebral disturbances, 76
caused by cysts in the orbit, 69
caused by dacryoadenitis, 74
caused by dermoid in the orbit, 70
caused by distended bursa of the trochlea, 69
caused by emphysema of the orbit, 66
caused by foreign body in the orbit, 67
caused by hemorrhage in the orbit, 66
caused by lymphomata, 70
caused by malignant growths, 70, 399
caused by Mikulicz's disease, 70
caused by orbital cellulitis, 73, 283
caused by osteoma in the orbit, 68
caused by periostitis of the orbit, 74
caused by sinusitis, 75
caused by swollen lacrimal gland, 70
caused by syphilis, 67
caused by tenonitis, 76
caused by tetanus, 74
caused by thrombosis of the cavernous sinus, 76, 343

Exophthalmos, caused by tuberculosis, 67
caused by tumor in the orbit, 68
caused by tumor of the optic nerve, 70
caused by vascular trouble in the orbit, 72
congenital, 64
following tenotomy, 64
from relaxation of the extrinsic muscles of the eye, 64
from unknown cause, 70
in exophthalmic goiter, 65
inflammatory, 73
intermittent, 73
physiological, 64
pulsating, 29, **71**
Exotropia, 82
Extrusion of the lens, 169
Exudate in the anterior chamber, 200, **201**, 219, 226, 295
Eye, foreign bodies in the, 302
injuries of the, 294
luxation of the, 67
penetrating wounds of the, 293, 301
sunken, 62
Eyeball, abnormal tension of the, 279
deviations of the, 62, **78**
displacements of the, 62
redness of the, 119
Eyeballs, normal relative positions of the, 78
Eyelash in the eye, 302
Eyelashes, 45
absence of the, 45
lice in the, 48
loss of color of the, 45
Eyespot of the nose, 462
Eyestrain, 112, 217, 438, **440**
a cause of urticaria, 29
and myopia, 452
due to abdominal troubles, 465
due to abnormal weakness of the accommodation, 450
due to astigmatism, 453
due to causes outside of the eye, 457
due to gastric trouble, 441, 466
due to heterophoria, 451, **454**
due to heterophoria, differential points concerning, 455
due to menstrual disorders, 465
due to presbyopia, 447
due to pressure in the nose, 452, **461**
due to refractive errors, 443
due to sinusitis, 463
due to spasm of the accommodation, 451
reflex, 457
simulated, 466

Facet, 167
Facial neuralgia, 281, 284, **438**
Facial paresis, 24, 52, 95, 178, **341**
False cataract, 262, 264
False coloboma, 214
False lenticonus, **252**, 266
False pterygium, 114
Familial defects, 13
Family history, 11, 12
Family idiocy, amaurotic, 363, **383**
Fascicular keratitis, 165
Favus of the lids, 50
Febrile herpes, 180, **181**
Fetal hyaloid artery, remains of the, 255, 259, **272**, **327**
Fibers, medullated nerve, 346, **356**, 431
Fibroma, 39, 115, 193
Field of fixation, monocular, 105
Field of vision, concentric contraction of the, 286, 381, 403, 419, **429**
contraction of the, 428
defects in the, 362, 370, 408, 419, **426**
displacement of the, 429
measurements of the, 427
normal, 426
sector shaped defects in the, **430**
tests of the, 426
Fields for color, 412, 427, **428**, 429
Filamentary keratitis, 184
Filariæ in the vitreous, 278
Filix mas, poisoning, 348, 414
Fistula, capillary lacrimal, 56
corneal, 169, 200, 292
lacrimal, 55
lacrimal, of the upper lid, 57
Fixation, monocular field of, 105
Flat detachment of the retina, 336, **401**
Flat sarcoma of the choroid, 406
Fluid cataract, 252
Fluid vitreous, 274
Fluorescin, 123, 163
Focal illumination, 7
Foerster's displacement of the field of vision, 429
Follicles, induced by conjunctivitis, **142**
induced by drugs, 143
Follicular affections of the conjunctiva, benign, **141**
Follicular conjunctivitis and its differentiation from trachoma, **144**
Folliculosis, school, **143**, 145
Foreign bodies in the eye, 293, 294, **302**
Foreign body, in the orbit, 67
in the upper transitional fold, **128**
in the vitreous, 278
localization in the eye by the X-rays, 304
on the surface of the eyeball, 24, 112, 120, 121, **122**, 124, 159, **162**, 309
ricochet of, after wounding an eye, 305
Foreshortening of the papilla, 360
Fovea centralis, 323
Fracture of the base of the skull, 19, 29, 117, 179, 330
Fracture of the wall of the orbit, 66, **117**
Frontal sinus, inflammation of the, 37, 59, 75, 119, 439, 463
Fugacious amaurosis, partial, 432
Fulminating glaucoma, 283
Fundus, abnormalities of the, 324
albinotic, 322
anæmia of the, 344
background of the, 322
diffuse pigmentation of the, 377, 385
general consideration of the, 314
hyperæmia of the, 309, **345**
large white spots in the, 356
pepper and salt, 377, **388**, 391, 392
red reflex of the, 8, 400
snuff colored, 377, 385, **388**, 391, 392
spots in the, 346
tessellated, 322
Fungi in corneal ulcers, **177**, 186, 190

Furuncle of the lid, 37
Fusiform cataract, 255

Gangrene of the lids, 35, 38
Gastric trouble, 283, 457, 466
Gelatinous scleritis, 196
General paresis, 21, 207, 239, 242, **244**, 245, 246, 331
Gerlier's disease, 22
Giant chalazion, 37
Gland, lacrimal. (See Lacrimal gland)
  preauricular, swelling of the, 58, 127, 399
  submaxillary lymphatic, swelling of the, 59
Glanders, 59, 74
Glands, lymphatic, 58
Glassblowers' cataract, 258, **261**
Glasses, correct adjustment of, 444
Glaucoma, 27, 119, 120, 124, 159, 183, 187, 194, 200, 209, 213, 215, 236, 238, 243, **280**, 333, 345, 348, 383, 420, 421, 431, 437, 468
  absolute, 195, 281, **291**
  acute inflammatory, 217, 218, **282**, 439
  acute inflammatory, prodromal symptoms of, 281
  chronic inflammatory, **284**, 439
  chronic inflammatory, symptoms of, 284
  fulminating, 283
  hemorrhages into the retina in, 290
  hemorrhagic, 200, 281, **290**
  infantile, 64, 158, 200, **287**, 399
  primary, 281
  prodromal symptoms of, 281
  secondary, 195, 217, 227, 230, 281, **288**, 396, 439
  secondary, after contusion of the eye, 289, 299
  secondary, causes of, 289
  simple, 285
  simple, intraocular tension in, 287
  symptoms of, 280
  with intraocular hemorrhages, 290
Glaucomatous cataract, 262
Glaucomatous cup or excavation, **281**, 318
Glaucomatous degeneration, 291
Glioma of the retina, 276, 386, 394, **395**, 405
Goiter, exophthalmic, 65
Gonorrhea, 56, 196, 221, 222, 338
Gonorrheal conjunctivitis, 58, **128**, 134, 172
  metastatic, 129
  of adults, 128
  of infants, **130**, 171
Gonorrheal iritis, 231
Gonorrheal ophthalmia, 130, 134
Gout, 196, 197, 222, 348, 378, 388, 393, 439
Gouty iritis, 232
Gouty retinitis, 378
Gradually developing pallor of the papilla, 330
Granular current, 369
Graves's disease. (See Exophthalmic goiter)
Greenish reflex of the lens, 262, 264, 267, 285
Groenouw's nodular degeneration of the cornea, 167
Group syphilide, 228

Guidance of the tears, 52
Gumma, of the choroid, 405
  of the conjunctiva, 152
  of the iris, 201, 228, **230**, 231, 237
  of the lids, 32, 34, 35, **36**
Gummatous basal meningitis, 20
Gummatous iritis, 230
Gummatous tarsitis, 36
Gunn's dots, **349**, 373
Gyrate atrophy of the choroid and retina, 382

Habit chorea, 24
Hæmophilia, 28, 66, 201, 348
Hallucinations, visual, 468
Halo, of the macula, **324**, 358
  of the papilla, 281, 285, **358**
  of the papilla, glaucomatous, 318, **358**
  of the papilla, senile, 358
Haziness of the papilla, 333
Headache, 281, **437**
  sick. (See Migraine)
Heart disease, 26, 380
Hemeralopia, 116, **380**, 385
Hemiachromatopsia, 433
Hemianopic pupillary reaction, **242**, 435
Hemianopsia, 21, 242, 331, 409, 419, 430, **432**
  binasal, 433, 434
  bitemporal, 433, 434
  distinctive features of, 433
  homonymous, 433, 434
  horizontal, 433, 434
  localization of brain lesions that cause, 434
  preservation of the macula in, 433
  quadrant, 434
  vertical, 433
Hemicrania, 438
Hemiopia, 433
Hemiplegia, 21
Hemorrhages, choroidal, **346**, 405
  in glaucoma, 290
  into the anterior chamber, 200
  into the lids, 28
  into the orbit, 29, **66**
  into the retina. (See Retinal hemorrhages)
  into the vitreous, **276**, 297
  into the vitreous, recurrent, **277**, 298, 366
  intracranial, 22, 340, 343
  preretinal, **277**, 347
  retinal, 298, **347**, 379
  subconjunctival, 117
Hemorrhagic diathesis, 29
Hemorrhagic glaucoma, 200, 281, **290**
Hemorrhagic retinitis, 378
Hereditary choroiditis, 392
Hereditary defects, 13, 213, 214, 250, 255, 256
Hereditary optic atrophy, 332
Hereditary syphilis, 188, 388, 391, 432
  Hutchinson's physiognomy of, 188
  Hutchinson's teeth symptomatic of, 188
  Hutchinson's triad symptomatic of, 188
Heredity, 107, 210, 288
Hernia cerebri, 38, 69, 72
Hernia of orbital fat, 32
Herpes, 32

Herpes zoster, 22, 24, **30**, 159, 181, 189, 439
ophthalmicus, 182
Herpes febrilis, or simplex, **150**, 180, **181**, 183, 185
Heterochromia, 204
Heterophoria, 101, **102**, 451, 454, 456, 466
a cause of eyestrain, 454
for near, 103
that fluctuates, 456, 466
Heterotropia, 82
Hippus, 241
History, family, 11, 12
taking, 11
Hole in the macula, **299**, **367**, 431
Homonymous hemianopsia, **433**, 434
Hordeolum, 37, 58, 119
externum, 37
internum, 37
Horizontal hemianopsia, 433
Horns, cutaneous, 39
Hutchinson's physiognomy of hereditary syphilis, 188
Hutchinson's syndrome, 17
Hutchinson's teeth, 188
Hutchinson's triad, 188
Hyaline degeneration, **152**, 168
Hyalitis, 275
Hyaloid artery, 255, 259, **272**, **327**
Hydatid cyst, 57, 69
Hydræmia, 26
Hydrocephalus, 242, 340, 343
Hydrophthalmos. (See Infantile glaucoma)
Hydrops of the third ventricle, 331
Hyperæmia, of the conjunctiva, 121
of the fundus, 309, **345**
of the fundus, active, 345
of the fundus, passive, 345
of the iris, **216**, 243
of the lids, 29, 46, 121
of the lids, active, 29
of the lids, passive, 29
of the margin of the lid, **47**, 121
of the papilla, 333
Hyperæsthesia of the cornea, 158
Hyperesophoria, 101
Hyperesotropia, 82
Hyperexophoria, 101
Hyperexotropia, 82
Hyperidrosis, 32
Hypermature senile cataract, 257, 265, **271**
Hypermetropia, 156, 296, **447**
a cause of convergent strabismus, 83, 85
latent, 449
manifest, 449
ratio of manifest to latent, 449, 451
total, 449
use of cyclopegics in, 451
Hyperphoria, 101
Hypertrophy, of the lacrimal gland, 57
of the skin of the lid, 32
Hypertropia, 82
Hyphæma, 200, 219, 226, 229, **295**
Hypophoria, 101
Hypophyseal disease, 22
Hypopyon, 201, 217, 219
Hypotropia, 82
Hysteria, 108, 240, 242, 419, 429, 459, 468
anæsthesia of the cornea in, 159
Hysteria, blepharospasm in, 25
Hysterical amblyopia, 412
Hysterical points of pressure, 25
Hysterical ptosis, 23

Idiocy, amaurotic family, 363, **383**
Illumination, focal, 7
oblique, 7
Imbalance of the ocular muscles, 121, 345. (See Heterophoria)
Immature cataract, 265, **268**
Immobility of the pupil, absolute, 208, **244**
amaurotic, 243
reflex, 208, 241, **245**
Impairment of vision by irregular astigmatism, 157
Impetigo, 30
Incipient cataract, 256, 265, **267**, 275
Indirect method, 9, 401
Indirect reaction of the pupil to light, 205
Indolent ulcer of the cornea, 166
Infancy, ulcers of the cornea in, 171
Infantile glaucoma, 64, 158, 200, **287**, 399
Infection, of anterior segment of wounded eye, 306
of a wounded eye, 119, **305**
of a wounded eye, first sign of, 305
Inferior conus, 326, 357, 358, **361**, 387
Infiltrate of the cornea, 161, **164**
Inflammation, sympathetic, 308, **310**
Inflammatory changes induced by wounds, 307
Inflammatory exophthalmos, 73
Inflammatory glaucoma, acute, 217, 218, **282**, 439
chronic, **284**, 439
Inflammatory white spots, 350
Influenza, 20, 79, 98, 190, 226, 235, 263, 283, 337, 348, 379, 394
Injection, conjunctival, 120, 124
episcleral, 120, 124
Injuries of the eye, 294
Insanity, 67, 238, 245, 468
Inspection of the conjunctiva, 111
Instillation of cycloplegics and of mydriatics for diagnostic purposes, 8, 125, 209, 218, 233, 238, 267, 289, 451, 452
Insufficiency of divergence a cause of convergent strabismus, 84
Insular sclerosis, 242
Intercalary staphyloma, 195
Intermittent exophthalmos, 73
Intermittent ophthalmomalacia, 293
Interstitial keratitis, 24, 162, **185**, 227, 288
Interval, in sympathetic inflammation, 312
in sympathetic irritation, 310
Intoxication a cause of nuclear paresis, 97
Inversion of the iris, **211**, 296
Inverted image, 9
Iodoform, toxic amblyopia from, 329, 413
Irideremia, 211
congenital, 212, 250
partial, 212, 214
total, 211
traumatic, 212, 296
Iridocyclitis, acute plastic, 119, 215, 218, 261, 283
serous, 216
sympathetic, 311
traumatic, 225, 307, 310

Iridodialysis, **211**, 296
Iridodonesis, 210
Iris, 202
  anteversion of the, 211
  artificial coloboma of the, 214
  atrophy of the, 227, **236**, 245
  bridge coloboma of the, 210, **214**
  coloboma of the, **213**, 250, 361
  color of the, 203
  contraction furrows or folds of the, 202
  corona of the, 202
  crypts of the, 202
  cysts of the, 201, 237
  false coloboma of the, 214
  hyperæmia of the, **216**, 243
  inversion of the, **211**, 296
  major circle of the, 203
  minor circle of the, 202
  partial coloboma of the, 213
  pigmented nævi of the, 204
  region of the sphincter of the, 202
  total coloboma of the, 213
  tuberculosis of the, 222, 229, **230**
  tumors of the, 219, 231, **236**
  zones of the, 202
Iritis, 27, 119, 124, 125, 182, 197, 201, **215**, 236, 243, 439
  ætiology of, 219
  aseptic purulent, 235
  chronic, 231, 236
  from anæmia, 234
  from infectious disease, 225
  from localized infections, 222
  gonorrheal, 231
  gouty, 232
  gummatous form of syphilitic, 230
  papular form of syphilitic, 226, **228**, 236
  parenchymatous, 224
  plastic, 215, **218**
  purulent, 204, **235**
  quiet, 216
  rheumatic, 222, **233**
  secondary, 215, **224**
  septic purulent, 235
  serous, 216
  superficial, 224, 226, 233
  syphilitic, 226
  traumatic, 225
  tuberculous, 216, 236
Irregular astigmatism, 141, **156**
Irritation, sympathetic, 309
Ischæmia, of the fundus, **345**, 346, 370, 413
  of the iris, 238

Keloid of the cornea, 193
Keratalgia, traumatic, 184
Keratitis, 120, 124, 182, 439
  annular, 186
  bullous, 181, **183**
  deep punctate, 191
  dendritic, 166, **180**
  disklike, 186
  e lagophthalmo, 23, 165, **177**, 180, 192
  fascicular, 165
  filamentary, 184
  interstitial, 24, 162, **185**, 227, 288
  interstitial, ulceration of the cornea in, 187
  interstitial, vascularization in, 185
  malarial, 181
Keratitis, marginal, 173
  neuroparalytic, 52, 159, 165, **179**, 182, 192
  parenchymatous. (See Keratitis, interstitial)
  phlyctenular, 24, 29, 43, 150, **172**, 467
  punctate interstitial, 186
  sclerosing, **191**, 195, 196, 197
  striped, 191
  superficial punctate, 185
Keratocele, 168
Keratoconjunctivitis, eczematous, scrofulous, or tuberculous, 172
Keratoconus, **157**, 200
Keratoglobus, **158**, 200
Keratomalacia, 178, 180, **192**

Labyrinthine troubles, 108
Laceration of the retina, 401
Lacrimal fistulæ, 55, 57
Lacrimal gland, cystic tumor of the, 57
  cysts of the, 57
  enlarged, 56
  exophthalmos caused by swollen, 70
  hypertrophy of the, 57
  mumps of the, 56
  subnormal secretion of the, 52
Lacrimal organs, **51**, 120, 122
Lacrimation, 51
Lagophthalmos, 41
Lamellar cataract, 253
Lamina cribrosa, 317, 329
Landry's disease, 22
Large white spots in the fundus, 356
Late papules of the iris, 228
Lead poisoning, 20, 97, 244, 289, 348, 375, 377
Lead, toxic amblyopia from, 329, 413
Lens, 247
  coloboma of the, 251
  consistency of the, 252
  dislocation of the, 209, **247**, 296, 301
  ectopia of the, 211, **249**, 257
  extrusion of the, 169, 249
  greenish reflex of the, 262, 264, 267, 285
  luxation of the, **247**, 296, 301
  luxation into the anterior chamber of the, 249, 296
  subluxation of the, **247**, 296, 301
Lenticonus, 251
  anterior, 251
  false, **252**, 266
  posterior, **251**, 255, 259
Leprosy, **50**, **154**, 159, 190, 197, 229, 236
Leucocythæmia, 40, 68, 201, 236, 276, 345, 352, 378
Leucocythæmic retinitis, 378
Leucoma, **166**, 264
  adherent, 169, 236
Leucosarcoma of the lids, 36
Lice in the eyelashes, 48
Lid margin, eczema of the, 48
  hyperæmia of the, 46
  inflammation of the, 48, 122
  pediculi of the, 48
  seborrhea of the, 47
Lids, 15
  abscess of the, 27, 37
  angioma of the, 31, 38, 39
  angiomegaly of the, 31

Lids, anthrax of the, 33
atrophy of the skin of the, 31
carbuncle of the, 38
chalazion of the, 35, **36**, 116, 122, 146, 152
chancre of the, 22, **33**, 35, 36, 58
chancroid of the, 34
cutaneous horns of the, 39
cyanosis of the, 29
dermatolysis of the, 31
dermoid of the, 38
discolorations of the, 29, **31**
ecchymoses of the, **28**, 117, **150**
emphysema of the, 28
epithelioma of the, 32, **35**, 36
eruptions on the, 29
eversion of the, 41, **111**
favus of the, 50
fibroma of the, 39
fugitive blackening of the, 29
fugitive œdema of the, 27, 60
gangrene of the, 35, 38
gumma of the, 32, 34, 35, **36**
hyperæmia of the, 29, 46, 121
hypertrophy of the skin of the, **32**
lipoma of the, 38
lupus of the, 32, 33
milia of the, 39
molluscum of the, 39
molluscum contagiosum of the, **39**
muscular tremor of the, 24
neurofibroma of the, 40, 288
œdema of the, 26
petechiæ of the, 29
pigmented nævi of the, **31**
sarcoma of the, 31, 35, **36**
secretory disorders of the skin of the, **32**
sores on the, 32
tumors of the, 35
ulcers of the, **32**, 33, 34
vaccine pustules on the, 32, 33, 58
warts of the, 39
wounds of the, 34, 294
Light, amblyopia from bright, 410
reaction of the pupils to, **205**, 243, 396
reflex arc, 206
reflexes, 10, **324**
streak of the retinal vessels, 320, **352**, 354
Lime burns, 154, 163
Lime deposits, in the conjunctiva, 153
Lipodermoid, 115
Lipoma, 32, 38, 115
Lithiasis of the conjunctiva, 153
Localization, of brain tumors, 341
of foreign bodies in the eye, 303, 304
Locomotor ataxia. (See Tabes dorsalis)
Lupus of the lids, 32, 33
Luxation, of cataract into the anterior chamber, 160
of the eye, 67
of the lens, **247**, 296, 301
Lymphangioma of the lid, 39
Lymphatic glands, **58**, 59
Lymphectasiæ, 115, 182
Lymphomata, 40, 70, 201, 236

Macula, 166
Macula lutea, **323**, 434
coloboma of the, 346, 357, 358, **363**, 387
Macula lutea, defects in the, **358**, 385
halo of the, **324**, 358
hole in the, **299**, **367**, 431
preservation of, in hemianopsia, 433
senile degeneration of the, 269, 372, 373, 390, **391**
Macular degeneration, 373, 377, **383**
Maculocerebral degeneration, 373, 377, **383**
Maddox rod test, 102
Major circle of the iris, 203
Malaria, 118, 180, 184, 190, 222, 225, 235, 338, 345, 348, 393, 411, 439
Malformation of the orbit or skull, 330, 338, 340
Malformations of the pupil, 208
Malignant growths in the orbit, 70
Malingering, 417
Malnutrition, 217
Marasmic children, 171, 192
Margin, of the papilla, 316
of the pupil, **202**, 295
Mariotte's blind spot, 431
Mature cataract, 265, **268**
Measles, 20, 74, 127, 132, 191, 207, 337, 394
Measurement of strabismus, 86
Measurements of the field of vision, 427
Mechanical ptosis, 17
Mechanism to maintain the normal relative positions of the eyeballs, 78
Medullated nerve fibres, 346, **356**, 431
Megrim. (See Migraine)
Meibomian glands, adenoma of the, **36**, 37
cysts of the, 35, 37
Melanosarcoma of lids, 36
Membrane, persistent pupillary, 203
Membranous cataract, 254, 257
Membranous conjunctivitis, 116, 117, **131**, 146
Meningitis, 20, 22, 25, 38, 108, 180, 241, 246, 263, 283, 338, 372, 394
Meningocele, 38, 69, 72
Menstrual disorders, 196, 217, 222, 348
Menstruation, 31, 119, 379
Mental diseases, 246
Metallic dots, 349
Metastatic choroiditis, 374
Metastatic gonorrheal conjunctivitis, 129
Methyl alcohol poisoning, 330, 332, **415**
Microphthalmos, 62
Migraine, 24, 25, 63, 119, 207, 283, **438**
ophthalmic, 22, **432**
Migratory ulcer of the cornea, 165, 174
Mikulicz's disease, 52, 70
Miliary tubercles in the choroid, 392, **393**
Milium, 39
Milky cataract, 257, 258
Mind blindness, 469
Miners' nystagmus, 109
Minor circle of the iris, 202
Molluscum contagiosum, 39
Molluscum simplex, 39
Monocular field of fixation, 105
Mooren's ulcer, 166, **174**
Morgagnian cataract, 257, **271**
Morphine poisoning, 208, 239, 246
Motor anomalies without deviation, **101**
Motor mechanism of the eyes, 78
Mucocele, of accessory sinus, 59, 60, 75
of the canaliculus, 53

Mucocele of the lacrimal sac, 54
Mucous patches on the conjunctiva, 152
Multiple neuritis, 22
Multiple sclerosis, 21, 99, 108, 207, 241, 245, 329, 331, 411, 430
Mumps, 56, 73, 379, 394
of the lacrimal gland, 56
Muscæ volitantes, 273
Muscle, absence of a, 94
Muscular paresis, **88**, 182, 341, 342
Bartel's test for, 78
differentiation from strabismus, 79
differentiation of lesions productive of, 94
due to cerebrospinal trouble, 94
due to a congenital anomaly, 93
due to lesion of a muscle, 94
due to orbital trouble, 94
due to rheumatism, 95
due to syphilis, 95
due to trichinosis, 94
nuclear, 21, 95, **96**
peripheral, 95
positions of the head in, 88, 89, 90
slight, 88
symptoms of, 88
torticollis in, 81, **90**
Muscular tremor, of lids, 24
Mushroom poisoning, 239
Mutton fat deposits on Descemet's membrane, 191, 217
Mydriasis, 205, **237**, 289
alternating, 242
induced by drugs, 237
reflex, 238
traumatic, 295
Mydriatics, use for diagnosis, 8, 125, 209, 216, 218, 233, 238, 248, 253, 267
Myelitis, diffuse, 108
chronic cerebral, 242
Myopia, 64, 85, 156, 263, 296, 347, 348, 359, 391, 446, 448, **452**
lenticular, 269
school, 359, 446
tone of ciliary muscle in, 453
Myosis, 205, 208, **239**, 246
paralytic, 238
spastic, 238
traumatic, 295
Myotonic convergence reaction, 207
Myxœdema, 27, 52
Myxoma of the cornea, 193

Nævi, pigmented, 31, 204, 237
Nævus of the conjunctiva, 113, 150
Nasal asthenopia, 452, 457, **461**
Nasal troubles, 121, 122, 439
Nebula, 166
Negative scotoma, 430
Nephritis, 26, 117, 119, 197, 276, 338, 339, 340, 348, 355, 371, 374, 375, 379, 391, 392, 393, 411
Nerve fibers, medullated, 346, **356**, 431
opaque, 346, **356**, 431
Nervous asthenopia, 457, **458**
Nervous disease, asthenopia and eyestrain from, 460
muscular paresis from, 94
Nervous exhaustion, 217, 458
Neuralgia, facial, 281, 284, **438**
supraorbital, 181, 215, 284, **438**, 439
Neuralgia, trigeminal, 24, 45, 52, 119, 181, 183, 283, 309, **438**
Neurasthenia, 207, 242, 459
Neuritic optic atrophy, **329**, 337
Neuritis, hypertrophic interstitial, 245
optic, 182, 329, 333, **335**
optic, diagnostic value of, 337
optic, differentiation from choked disk, 335
optic, in one eye alone, 339
Neurofibroma of lids, 40, 288
Neuron, centrifugal, **206**, 208
centripetal, 206
Neuroparalytic keratitis, 52, 159, 165, **179**, 182, 192
Neuroretinitis, 275, 357, 363, 371, **431**
sympathetic, 312, 339
Nictitation, 24
Nitrobenzene poisoning, 348, 414
Nodular degeneration of the cornea, **167**
Noma, 35
Normal field of vision, 426
Normal pupil, 201, 204
Normal relative position of the eyeballs, 78
Nose, eyespot of the, 462
Nuclear ophthalmoplegia, chronic, 98
Nuclear paresis, 21, 95, **96**
acquired, 96
congenital, 96
due to diabetes, 98
due to diseases of the central nervous system, 97
due to exophthalmic goiter, 99
due to infectious disease, 97
due to toxic causes, 97
Nystagmic twitchings, **107**, 341
Nystagmus, 101, **106**, 241, 250, 383
a compensatory phenomenon, 107
acquired, 107
acquired, in nervous diseases, 108
congenital, 107
hereditary predisposition to, 107
miners', 109
occupational, 108
of aural origin, 108
unilateral, 107, 109
voluntary, 107

Objective symptoms, 6
Oblique illumination, 7
Obliteration of the anterior chamber, 199
Observations to be made in strabismus, 86
Occluded pupil, 209, 227
Occlusion of a branch of the central artery, 370
Occlusion of the central artery, 332, 336, 345, 363, **368**, 383
Occupational nystagmus, 108
Occupational ptosis, 25
Œdema, of the conjunctiva, 118
of the lids, 26
of the lids, fugitive or recurrent, 27, 60
of the lids, in dacryocystitis, 27
of the lids, inflammatory, 27
of the lids, in inflammation of the accessory sinuses, 27
of the lids, malignant, 28
of the lids, noninflammatory, 26
of the lids, solid, 28, 30
of the retina, 345, 346, **366**, 401

Œdema of the retina, traumatic, **298**, 336, 363, **367**
Oil, aniline, toxic amblyopia from, 413
of wintergreen, toxic amblyopia from, 414
Onyx, 190
Opacities, in the cornea, 161
in the vitreous, 268
Opacity, Berlin's. (See Traumatic œdema of the retina)
Opaque nerve fibers, 346, **356**, 431
Ophthalmia, neonatorum, 130
nodosa, **127**, 229
sympathetic, 45, 217, 225, 231, 292, **301**, **308**
Ophthalmic migraine, 22, **432**
Ophthalmomalacia, intermittent, 293
Ophthalmoplegia, chronic nuclear, 98
chronic progressive, 16, 21, **98**
external, 92
internal, 92
total, 91
Ophthalmoscopy, **7**, **400**
Opium poisoning, 208, 239, 246
Optical agnosia, 469
Optical aphasia, 469
Optic atrophy, 182, **327**, 416, **421**
choroiditic, 389
hereditary, 332
neuritic, **329**, **337**
partial, 327
retinitic, 332
simple, 329
total, 327
Optic nerve, disease of the papillomacular bundle of fibers of the, **328**, 376, 431
sheath, coloboma of the, 318, **326**, 358
tumors of the, 70
Optic neuritis, 182, 329, 333, **335**
diagnostic value of, 337
differentiation from choked disk, **335**
in one eye alone, 339
Orbit, emphysema of the, 66
hemorrhage into the, 29, **66**
malformation of the, 338
tumors in the, 59, **68**, 119
Orbital cellulitis, 27, 37, 56, 60, **73**, 119, 283, 339, 380
Orbital periostitis, 27, 37, 73, **74**, 75, 119, 339
Orbital trouble, a cause of muscular paresis, 94
Orthophoria, 101
Osteoma of the orbit, 59, 68
Otitis media, 333, 338, 343, 366, 439
Oval pupil, 209
Overwork, 217, 416
Oxycephalus, 330

Pachymeningitis, hemorrhagic, 340
Pain, 5, 437
Pallor of the papilla, gradually developing, 330
sudden, 330
Pannus, 139, 147, 151, 161
carnosus, 140
crassus, 140
siccus, 140, 141
tenuis, 140
trachomatous, **139**, 148
vasculosus, 140
Panophthalmitis, 27, 73, 74, 182, 225, 235, **306**, 307, 374, 394
Papilla, **315**, **326**
color of the, 315
defects about the, 358
excavations of the, 281, 317, **318**
gradually developing pallor of the, **330**
halo of the, 281, 285, 318, **358**
margin of the, 316
pigmentation of the, 321
pulsation of vessels on the, **321**, **354**
redness of the, 333
size of the, 315
sudden pallor of the, **330**
vessels of the, 319
Papilloma, of the conjunctiva, 115
of the cornea, 193
Papillomacular bundle, disease of the, **328**, 376, 431
Papular form of syphilitic iritis, 226, **228**, 236
Papules, early, 228
late, 228
Paracentral scotoma, 430
Paralysis, after diphtheria, 244, **246**
agitans, 99, 108
or paresis, conjugate, 92, **99**
of convergence, 92, **100**
of divergence, **93**, 100
Paralytic ectropion, 42
Parasites in the vitreous, 278
Parenchymatous iritis, 224
Parenchymatous keratitis. (See Interstitial keratitis)
Paresis, facial, 24, 52, 95, 178, 341
general, 21, 207, 239, 242, 244, 245, 246, 331
muscular. (See Muscular paresis)
nuclear. (See Nuclear paresis)
of external rectus, 88, 89, 98
of inferior oblique, 90
of inferior rectus, 90
of internal rectus, 88, 89
of muscles supplied by the third nerve, 91, 95, 96, 241
of superior oblique, 90, 94
of superior rectus, 90
peripheral, 95
supranuclear, 95
Parinaud's conjunctivitis, 58, 146, **148**
Partial fugacious amaurosis, 432
Partial irideremia, 212, 214
Partial staphyloma, 170
Patch, salmon, 161, 186
Pediculi of the lids, 48
Pemphigus, 31, 44, 116, 117, 134, 146, **151**
Penetrating wounds of the eye, 293, **301**
Pepper and salt fundus, 377, **388**, 391, 392
Perforation of an ulcer of the cornea and its consequences, 168
Perimeter, 87, **427**
Periostitis of the walls and margin of the orbit, 27, 37, 73, **74**, 75, 119, 339
Peripheral paresis, 95
Peripheral scotoma, 430
Peripheral zone, 203
Pernicious anæmia, 345, 352, 378, 379
Persistent hyaloid artery, 255, 259, **272**, **327**

Persistent pupillary membrane, 203
Petechiæ of the lids, 29
Phlyctenular conjunctivitis, 29, 58, 136, **149**, 197
Phlyctenular keratitis, 24, 29, 43, 150, **172**, 467
Phlyctenules, 112, 135, 136, **149**, 181, 197
Phorometer, 102
Phosphorus poisoning, 348, 379, 413
Phthisis bulbi, 227, **292**, 307
Physiological cup or excavation of the papilla, **317**, 318
Physiological protrusion of the eyeball, 64
Pigmentary degeneration of the retina, 255, 259, 263, 332, 350, **380**, 385, 431
  without pigment, 381
Pigmentation, of the cornea, 159
  of the papilla, 321
  of the sclera, 194
Pigmented nævi, 31, 204, 237
Pigment ring, 316
Pinguecula, 112
Plastic iritis, 215, **218**
Pneumococcal ulcer of the cornea, 175
Pneumonia, 20, 222, 225, 235, 242, 337, 394
Poison ivy, 119
Polar cataract, anterior, 169, **256**, 259, 260
  posterior, **255**, 259, 383
Polioencephalitis, 97
Poliomyelitis, 22, 97, 242
Polycoria, 210
Polymyositis, 22
Polyneuritis, 338
Polyp of the conjunctiva, 115
Pomegranate, toxic amblyopia from, 413
Poor choroid, 323
Positions of the eyeballs, normal relative, 78
Positions of the head in muscular paresis, 88, 89, 90
Positive scotoma, 430
Postdiphtheritic paralysis, 244, 246
Posterior accessory sinuses, inflammation of the, 61, 76, 329, 339, 340
Posterior ethmoidal cells, symptoms of inflammation of the, 61, **76**, 339, 340, 431
Posterior lenticonus, **251**, 255, 259
Posterior polar cataract, **255**, 259, 383
Posterior staphyloma, 195, 346, 357, 358, **359**, 387, 431
  foreshortening of the papilla in, 360
Posterior synechia, total, 209, 261
Posterior synechiæ, 209
Potassium chlorate poisoning, 348, 414
Powder stains, 31, 194
Preauricular gland, swelling of the, 58, 127, 399
Pregnancy, 31, 52, 72, 108, 276, 372, 393
  albuminic retinitis of, **375**, 393
Preretinal hemorrhage, **277**, 347
Presbyopia, 446
Preservation of the macula in hemianopsia, 433
Primary deviation, 80
Primary glaucoma, 281
Prism convergence, 104
Prism divergence, 104
Prism tests, 87, 102, 104
Prodromal symptoms of glaucoma, 281
Progressive facial hemiatrophy, 32
Progressive muscular paralysis, nuclear paresis in, 99
Projection, 269
Proliferating retinitis, 277, 298, 357, 363, **365**
Proliferation of connective tissue into the vitreous, 307, 365
Prophylaxis, of gonorrheal conjunctivitis of infants, 131
  of sympathetic inflammation, 312
Prostatitis, 232
Pseudoglioma, 276, 395, **398**
Pseudoleucocythæmia, 236
Pseudoneuritis, **334**, 336
Pseudopterygium, 114
Pseudoptosis, 18, 25, 26
Psychical blindness, 467
Pterygium, 112, **113**, 122
  false, 114
Ptomaine poisoning, 20, 97, 238, 244, **289**, 411
Ptosis, 16
  acquired, 17
  congenital, 16
  from cerebrospinal lesions, 19
  from lesions of the sympathetic nerve, **22**
  hysterical, 23
  in acute infectious diseases, 20
  in infants, 19
  in syphilis, 20
  isolated bilateral, 18
  mechanical, 17
  senile, 18
  traumatic, 18
Puerperal fever, 73, 116, 192, 394
Pulsating exophthalmos, 29, **71**
Pulsation of vessels on the papilla, **321**, **354**
Punctata albescens, **349**, 382
Punctate cataract, **256**, 270
Punctate interstitial keratitis, 186
Punctate keratitis, deep, 191
  superficial, 185
Punctum lacrimalium, closure of the, 53
Pupil, absolute immobility of the, 208, **244**
  amaurotic immobility of the, 243
  Argyll-Robertson, 245
  attention reflex of the, 207
  cortical reflex of the, 207
  crater, 198, **209**, 227, 231, 261
  excluded, 209, 227
  hemianopic reaction of the, **242**, **435**
  malformations of the, 208
  myotonic reaction of the, 207
  normal, 201, **204**
  occluded, 209, 227
  oval, 209
  reaction of, to accommodation, 206
  reaction of, to closure of the lids, 207
  reaction of, to convergence, 206
  reactions of, to light, **205**, 243, 396
  reflex contractions of the, 205
  reflex dilatation of the, 208
  reflex immobility of the, 208, 241, **245**
  secluded, 209
  slow reaction of, to light, 206, **243**
Pupillary membrane, persistent, 203
Pupillary margin, **202**, 295
Pupillary zone, 202
Pupils, symptomatology of the, 237

Purpura hemorrhagica, 29, 31, 118, 201, 348
Purulent choroiditis, **394**, 397
Purulent iritis, 204, **235**
Pus, sterile, 201, 235

Quiet iritis, 216
Quincke's disease, 27
Quinine, toxic amblyopia from, 330, **332**, 345, 413, **414**

Rainbow-tinted halos about lights, 282, **420**
Reaction, hemianopic pupillary, **242**, 435
of pupils to light, slow, 206, **243**
Reactions, of the pupil to light, **205**, 243, 396
of the pupils, 205
Recurrent chronic cyclitis, 217
Recurrent erosion, 163, 184
Recurrent fever, 348
Recurrent hemorrhages into the vitreous, **277**, 298, 366
Redness of the eyeball, 119
Redness of the papilla, 333
Red reflex of the fundus, 8, 400
Red spots, 346
Reflex amblyopia, 411
Reflex asthenopia, 457
Reflex contractions of the pupils, 205
Reflex dilatation of the pupils, 208
Reflex eyestrain, 457
Reflex immobility of the pupils, 208, 241, **245**
Reflex, red, 8, 400
Reflex ring, Weiss's, **324**, 360
Reflexes, light, 10, **324**
Refraction, errors of, 121, 345, 443
eyestrain due to errors of, 443
varying effects produced by errors of, 444
Region of the sphincter of the iris, 202
Regular astigmatism, 156
Relative positions of the eyeballs, normal, 78
Remains of the fetal hyaloid artery, 255, 259, **272**, **327**
Remains of the fetal pupillary membrane, 203
Retention cysts, 39, 57
Retina and choroid, 344
gyrate atrophy of the, 382
Retina, angioid streaks in the, 353
apoplexy of the, 379
blood vessels of the. (See Retinal vessels)
detachment of the. (See Detachment of the retina)
bullous detachment of the, 396, 401
flat detachment of the, 336, 401
glioma of the, 276, 386, 394, **395**, 405
hemorrhages into the. (See Retinal hemorrhages)
laceration of the, 401
œdema of the, 345, 346, **366**, 401
pigmentary degeneration of the, 255, 259, 263, 332, 350, **380**, 385, 431
traumatic œdema of the, **298**, 336, 363, **367**
Retinal hemorrhages, 298, **347**, 379
spontaneous, 348
toxic, 348
traumatic, 347
Retinal vessels, **319**, **350**
beaded appearance of the, 355
color of the, 352
light streak of the, 320, **352**, 354
relative sizes of the, 320, 351
sclerosis of the, 354
sheathing of the, **351**, 354
size of the, 320
tortuosity of the, 320, **352**, 354
varicosities of the, 353
walls of the, 320, **352**
Retinitic optic atrophy, 332
Retinitis, 363, **370**, 386
albuminuric, 374
albuminuric, of pregnancy, **375**, 393
anæmic, 378
causes of, 371
central, 366, 370, **372**, 385, 390
circinate, 386
diabetic, 376
effect of on vision, 371
gouty, 378
hemorrhagic, 378
leucocythæmic, 378
pigmentosa, 381
proliferating, 277, 298, 357, 363, **365**
punctata albescens, 350, 382, 386
saturnine, 377
septic, 374
striate, 353
syphilitic, 376
Retinochoroiditis juxtapapillaris, **390**, **430**
Retraction of eyeball into the orbit, 63
Rheumatic iritis, 222, **233**
Rheumatism, 20, 95, 190, 196, 197, 219, 221, 222, 231, 233, 338, 393
Rhinophyma, 32
Ribbon-shaped degeneration of the cornea, 167
Rickets, 190, 263
Ricochet of foreign body after wounding the eye, 305
Ring, connective tissue, 316
pigment, 316
scleral, 316
scotoma, 430, **431**
Weiss-Otto shadow, 324, **360**
Weiss's reflex, **324**, 360
Rodent ulcer of the lid, 35
Rupture of the choroid, 299, **364**, 387
direct, **299**, 364
indirect, **299**, 364
Rupture of the eyeball, 300

Salicylic acid, toxic amblyopia from, 345, 414
Salmon patch, 161, 186
Santonin, 421
Sarcoma, of the choroid, 394, 397, 399, **405**
of the choroid, flat, 406
of the conjunctiva, 113, 114, 115, 136
of the cornea, 193
of the iris, 201, 204, 230, 231, 237
of the lid, 31, 35, **36**
of the orbit, 70
Saturnine retinitis, 377
Scammony, toxic amblyopia from, 413
Scarlet fever, 45, 74, 116, 132, 191, 192, 263, 337, 394
School folliculosis, **143**, 145

School myopia, 359, 446
Scientific nomenclature of strabismus, 82
Scintillating scotoma, 432
Sclera, 194
  pigmentation of the, 194
  staphyloma of the, 194
Scleral ring, 316
Scleritis, 120, 182, 194, **195**
  annular, 196
  brawny, 196
  deep, 192, 195
  gelatinous, 196
  superficial, 197
Scleroderma, 32
Sclerosing keratitis, **191**, 195, 196, 197
Sclerosis, of the choroid, 346, **357**, 358, 361, 363, 364, 385
  of the retinal vessels, 354
Scotoma, 61, 386, 409, **430**
  annular, 430
  central, **298**, **328**, 339, 367, 376, 390, **430**, **431**
  central color, 411, 415, 426, 427
  color, test for, 427
  negative, 430
  paracentral, 430
  peripheral, 430
  positive, 430
  ring, 430, **431**
  scintillating, 432
Scrofula, 172, 181, 190
Scurvy, 29, 118, 348
Seborrhea, 47
  oleosa, 48
  sicca, 47
Secluded pupil, 209
Secondary deviation, 80
Secondary glaucoma, 195, 217, 227, 230, 281, **288**, 396, 439
  after contusion, 299
  causes of, 289
Secondary iritis, 215, **224**
Sector-shaped defects in the field of vision, 409, **430**
Semilunar fold, 110, **112**, 142
Senile cataract, 252, 262, **264**
Senile degeneration of the macula, 269, 372, 373, 390, **391**
Senile ectropion, 42
Senile entropion, 43
Senile ptosis, 18
Sense, affections of the color, 420
Sepsis, 29, 372
Septicæmia, 98, 374, 394
Septic retinitis, 374
Serous cyclitis, 216
Serous iridocyclitis, 216
Serous iritis, 216
Serpiginous syphilide, of the lids, 32, 33
Serpiginous ulcer of the cornea, 165, **174**, 177
Shadow ring, Weiss-Otto, 324, **360**
Shallow anterior chamber, 199
Sheathing of the retinal vessels, **351**, 354
Sheath of the optic nerve, coloboma of the, 318, **326**, 358
Sick headache. (See Migraine)
Siderosis, of the cornea, 160
  of the iris, 204, 236
Siliquose cataract, 254, **257**, 258, 271
Simple glaucoma, 285
  subjective symptoms of, 286
  tension in, 287
Simple optic atrophy, 329
  causes of, 330
Simple ulcer of the cornea, 164
Simulation, of amblyopia, 418
  of blindness, 417
  of defects in the field of vision, 419
  of eyestrain, 466
Sinuses, accessory, empyemas of the various, 37, 59, 60, 61, 74, 75, 431, 463
Sinusitis, eyestrain from, 463
  iritis from, 221
Sinus thrombosis, 76, 333, 338, 340
Size of the papilla, 315
Size of the retinal vessels, 320
Slowness of reaction of the pupil to light, 206, **243**
Small-pox, 32, 191
Small white spots, 348
Snake bite, 97, 348
Snow blindness, 154
Snuff colored fundus, 377, 385, **388**, 391, 392
Softening of the brain, 19, 22
Solid œdema, 28, 30
Solitary tubercle, of the choroid, 394, 397
  of the iris, 230, 231
Sores on the lids, 32
Sparkling synchysis, 275
Spasm of the accommodation, 296, **451**, 452
  simulating myopia, 451, 452
Spasmus nutans, 108
Spastic ectropion, 42
Spastic entropion, 44
Sphenoidal sinus, symptoms of inflammation of the, 61, **76**, 339, 340
Spinal cord, disease of the, 238
Spindle cataract, 255
Spontaneous subconjunctival hemorrhages, 117
Sporotrichosis, 33, 149
Spot, blind, 431
  cherry red, **368**, 383
Spots, black, **346**, 381, 387
  inflammatory white, 350
  in the fundus, 346
  large white, 356
  on the conjunctiva, 113
  red, 346
  small white, 348
  white, 382
Squirrel plague conjunctivitis, 127
Staphyloma, anterior, 178, 195
  ciliary, 195
  equatorial, 195
  intercalary, 195
  of the cornea, 169, **170**, 193
  of the cornea, partial, 170
  of the cornea, total, 170
  of the sclera, 194
  posterior, 195, 346, 357, 358, **359**, 387, 431
Starvation, 116, 192
Stellate figure of the macula, 375
Sterile pus, 201, 235
Strabismometer, 87
Strabismus, 79, **82**
  and the angle γ, 87

Strabismus, convergent, **82**. (See Convergent strabismus)
differentiation from muscular paresis, 79
divergent, 79, 82, **85**, 341
due to cicatricial adhesions, 82
measurement of, 86
observations to be made in, 86
scientific nomenclature of, 82
vertical, 82
Streaks, angioid, in the retina, 353
light, of the retinal vessels, 320, **352**, 354
Streptococcus in membranous conjunctivitis, 132, 134
Striate retinitis, 353
Striped keratitis, **191**
Styes, 37
Subcapsular cortical cataract, 267
Subconjunctival hemorrhage, 117
Subcutaneous tuberculin test, 147, 189, 231
Subjective symptoms, 6
of chronic inflammatory glaucoma, 284
of incipient senile cataract, 265
of simple glaucoma, 286
Subluxation of the lens, **247**, 296, 301
Subnormal secretion of the lacrimal gland, 52
Subnormal tension, 183, **187**, 239, 274, **291**, 299, 301
after contusion, 292, 299
Sudden pallor of the papilla, 330
Sunken eye, 62
Superficial iritis, 224, 226, 233
Superficial punctate keratitis, 185
Supertraction, 316, 360
crescent, **316**, 336
Supranuclear paresis, 95
Supraorbital neuralgia, **181**, 215, 284, **438**, 439
Sursumduction, 104
Sursumvergence, 104
Sweet's apparatus for locating foreign bodies in the eye or the orbit, 304
Sycosis, coccogenes, 49
parasitica, 49
Symblepharon, 82, **116**, **151**, 154
Sympathetic inflammation, 308, **310**
prophylaxis of, 312
Sympathetic iridocyclitis, 311
Sympathetic irritation, 308, **309**
Sympathetic nerve, enophthalmos in lesions of the, 63
exophthalmos in lesions of the, 65
irritation of the, 238
paralysis of the, 121, 239, 240, 291
ptosis in lesions of the, 22
Sympathetic neuroretinitis, 312, 339
Sympathetic ophthalmia, 45, 217, 225, 231, 292, 301, **308**
Symptomatology of the pupils, 237
Symptoms, 5
objective, 6
ocular, of inflammation of the posterior ethmoidal cells, 61, **76**, 339, 340, 431
ocular, of inflammation of the sphenoidal sinus, 61, **76**, 339, 340
ocular, of thrombosis of the cavernous sinus, 73, 76, 343
of asthenopia, 440
of eyestrain, 441
Symptoms, subjective, 6
Synchysis scintillans, 275
Syndrome of Hutchinson, 17
Synechiæ, anterior, 169
posterior, 209
Syphilis, 20, 45, 67, 74, 95, **97**, 99, **117**, 146, 180, 181, 188, 196, 197, 219, 221, 222, 276, 289, 338, 354, 355, 366, 371, 388, 391, 392, 411, 432, 439
cerebral, 241, 244, 245, 246, 331
hereditary, 188, 388, 391, 432
Hutchinson's physiognomy of hereditary, 188
Hutchinson's teeth in hereditary, 188
Hutchinson's triad in hereditary, 188
Syphilitic iritis, 226
Syphilitic lesions of the conjunctiva, **151**
Syphilitic periostitis, 74
Syphilitic retinitis, 376
Syringomyelia, 99, 108, 245

Tabes dorsalis, 21, 52, 95, 98, 108, 159, 207, 239, 242, 244, 245, 246, 329, 330, 332, 430, 460
Table to aid in locating paretic muscles, 90
Taking the history, 11
Tape measure test, 86
Tarsitis, gummatous, 36
tuberculous, 35, 37
Tay's choroiditis, 373, 390
Tea, toxic amblyopia from, 415
Tears, guidance of the, 52
Teeth, apical infections of the, 222
carious, 411
Tenonitis, 73
purulent, 76
serous, 76
Tension of the eyeball, 279
in simple glaucoma, 287
subnormal, 183, 187, 239, 274, **291**, 299, 301
Tessellated fundus, 322
Test, Calmette's, 189, 230
cover, 103
for central color scotoma, 427
Maddox rod, 102
prism, 87, 102, 104
Wernicke's, 242, 435
Tests, for nasal asthenopia, 462
of the tension of the eyeball, 279
of the visual field, 426
Tetanus, exophthalmos from, 74
Third nerve paresis, 91, 95, 96, 238, 241
Thrombophlebitis of the facial veins, 73
Thrombosis, of the cavernous sinus, 73, 76, 343
of the central vein, 379
of the longitudinal sinus, 77, 343
of the orbital veins, 38, 73
Tiger choroid, 322
Tobacco and alcohol, toxic amblyopia from, 328, 411, 413, **415**
Tone of the ciliary muscle, 448, 449
in myopia, 453
Tonometer, 219, **279**, 280, 439
Tonsillar infection, 221, 222
Tonsillitis, 73, 74, 394
Torsion, 106
Torticollis from muscular paresis, 81, **90**

Tortuosity of the retinal vessels, 320, 352, 354
physiological, 320
Total cataract, **254**, 259
Total coloboma of the iris, **213**
Total irideremia, 211
Total staphyloma, 170
Toxæmia, 208
Toxic amblyopia, 413
from alcohol and tobacco, 328, 411, 413, **415**
Toxic asthenopia, 121, 460
Trachoma, 43, 44, 116, 117, 135, **136**, 141, 143, 144, 145, 147, 148, 172
acute, 139
cicatricial, 139, 141
contagiousness of, 136
corneal ulcer of, 166
early stage of, 136
mixed, 138
granules of, 137
pannus of, 139
papillary, 138
proliferation of papillæ in, 136, 138
sequelæ of, 140
Transillumination of the eye, 402, 404, 406
Traumatic amblyopia, 410
Traumatic cataract, **257**, 262, 297
Traumatic enophthalmos, 63
Traumatic irideremia, 212, 296
Traumatic iritis, cyclitis, and iridocyclitis, **225**, 307, 310
Traumatic keratalgia, 184
Traumatic mydriasis, 295
Traumatic myosis, 295
Traumatic neurosis, 25, 419, 429, 468
Traumatic œdema of the retina, **298**, 336, 363, **367**
Traumatic ptosis, 18
Traumatic ulcer of the lids, 34
Traumatism, 18, 27, 28, 32, 66, 74, 97, 117, 118, 127, 172, 175, 177, 186, 189, 201, 207, 211, 255, 369, 371, 391
Tremor of the lids, 24
Trichiasis, **46**, 140, **151**
Trichinosis a cause of muscular paresis, 94
Trigeminal neuralgia, 24, 45, 52, 119, 181, 183, 283, 309, **438**
Tropometer, 105
Tubercles in the choroid, conglomerate, 394, 397
miliary, 392, **393**
solitary, 394, 397
Tubercles in the fundus, 338, 386, 392, 393, 394, 396, 397
Tubercles in the iris, 201, 230, 231, 237
Tuberculin tests, 147, 189, 230
Tuberculoma of the choroid, 394, 399, 405
Tuberculosis, 97, 99, 172, 181, 191, 196, 197, 221, 338, 363, 372, 388, 391, 392, 397
and interstitial keratitis, 189
and phlyctenular keratitis, 172
anisocoria an early symptom of, 240
bovine, 149
of the choroid, 393
of the conjunctiva, 28, 58, 128, 136, **146**, 152
of the iris, 222, 229, **230**
Tuberculosis of the orbit, 67, 74
Tuberculous choroiditis, 393
Tuberculous iritis, 216, 236
Tuberculous periostitis, 75
Tuberculous tarsitis, 35, 37
Tumors, intraocular, 193, 200, 201
of the brain, 22, 25, 239, 331, 375, **434**
of the brain, choked disk in, 340
of the brain, localization of, 341
of the brain, symptoms of, 340
of the choroid, 394, **405**
of the choroid, differentiation from detachment of the retina, 402
Tumors, of the conjunctiva, 114
of the cornea, 170, **192**
of the iris, 219, 231, **236**
of the lids, 35
of the optic nerve, 70
of the orbit, 59, **68**, 119
Typhoid fever, 20, 45, 55, 74, 98, 116, 191, 192, 222, 225, 235, 263, 337, 348, 372, 394

Uhthoff's degeneration of the cornea, 168
Ulcers of the conjunctiva, 58, 146, 152
Ulcers of the cornea, 162, **164**, 187, 215
caused by the bacillus pyocyaneus, 176
caused by fungi, **177**, 186, 190
crescentic, 165, 173
deep purulent, 165, 177
diplobacillus, 175, 176
in childhood, 172
indolent, 166
in infancy, 171
in keratitis e lagophthalmo, 165
in middle and old age, 174
in neuroparalytic keratitis, 165
met with at all ages, 177
migratory, 165, 174
Mooren's, 166, **174**
on the posterior surface, 166
perforation of, **168**, 293
pneumococcal, 175
serpiginous, 165, **174**, 177
simple, 164
trachomatous, 166
traumatic, 177
Ulcers of the lids, 32
diphtheritic, 34
rodent, 35
traumatic, 34
tuberculous, 32, 33
Unilateral nystagmus, **107**, 109
Upright image, 10
Uræmia, 20, 238, 239
Uric acid diathesis, **121**, **122**, **153**, 233, 234, 259
Urticaria, caused by eyestrain, 29
Use of mydriatics and cycloplegics for diagnostic purposes, 8, 125, 209, 218, 233, 238, 267, 289, 451, 452
Uterine troubles, 217, 458
Uvea, ectropion of the, 202
Uveitis, 187, 261, 262, 274, 292, 388

Vaccine pustule on the lids, 32, 33, 58
Variation in symptoms produced by any disease, 14
Varieties of corneal ulcers, 164

Vascularity of the cornea in interstitial keratitis, 185
Vascular troubles in the orbit, 72
Vein, thrombosis of the central, 379
Veins, thrombosis of the orbital, 38, 73
Venous circulation, obstruction to the, 29
Venous pulse, 321
Vernal catarrh, 134
  bulbar, 135
  mixed, 135
  tarsal, 134
Vertical hemianopsia, 433
Vertical strabismus, 82
Vessels, choroidal, 322
  cilioretinal, **321**, 369
  of the papilla and retina, 319
  retinal. (See Retinal vessels)
Vision, color, 421
  impairment of by irregular astigmatism, 157
  in choked disk, **335**, 343
  measurement of the, 443
Visual amnesia, 469
Visual aphasia, 469
Visual field, contraction of the, 286, 381, 403, 419, **428**
  defects in the, 362, 370, 408, 419, **426**
  displacement of the, 429
  measurements of the, 427
  normal, 426
  sectorshaped defects in the, 409, **430**
  tests of the, 426
Visual hallucinations, 468
Vitiligo, 31
Vitreous, 272
  abscess of the, 275, **307**, 397
  cysticercus in the, 278, 397
  filariæ in the, 278
  fluid, 274
  foreign bodies and parasites in the, **278**
  hazy, 275
  hemorrhage into the, **276**, 297
  opacities in the, 268
  parasites in the, 278
  recurrent hemorrhages into the, **277**, 298, 366
Voluntary nystagmus, **107**
Von Pirquet's test, **147**, **173**, **189**, **230**

Walls of the retinal vessels, 320, **352**
Warts on the lids, 39
Weakness of the accommodation, 309, 450
Weiss-Otto shadow ring, 324, **360**
Weiss's reflex ring, **324**, 360
Wernicke's test of the pupillary reaction, 242, 435
White spots in the fundus, 382
  inflammatory, 350
  large, 356
  small, 348
Whooping cough, 28, 117, 337, 345
Winking, 23
  slow, 23
Wintergreen, toxic amblyopia from oil of, 414
Wood alcohol poisoning, 330, 332, **415**
Word blindness, 469
Worms, intestinal, 238
Wounded eye, infection of a, 305
  iridocyclitis of a, 307
Wounds, inflammatory changes induced by, 307
  of the ciliary body, 308
  of the cornea, 162
  of the eye, 293, 294, 301
  of the lid, 34, 294
  penetrating, 293, **301**
Wrinkling of Descemet's membrane, **191**

Xanthelasma, or xanthoma, 31, 39
Xeroderma pigmentosum, 31
Xerophthalmos, 151
Xerosis, **116**, 140, 172, 192
  cicatricial, 117
  epithelial, 116
X-rays, 222, 258, 261, 304
  to detect foreign bodies in the eye, **304**

Zones of the iris, 202
Zonular cataract, **253**, 259
Zygomatic fossa, abscess in the, 61, **74**, **75**

28.A.412.
Diagnosis from ocular symptoms.1917
Countway Library
AGQ8840
3 2044 045 058 385

28.A.412.
Diagnosis from ocular symptoms.1917
Countway Library
AGQ8840
3 2044 045 058 385